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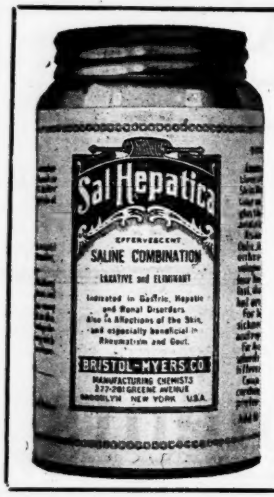

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MUSCLE ATROPHY.[1]

By **A. Juett, B.M., B.Ch. (Oxon.)**,
Perth.

It is impossible to deal with so large a subject as muscle atrophy in a short paper. My attempt will, therefore, resolve itself into an imperfect summary of some of the problems of the wasted voluntary muscle.

Structure.

The unit of action is the muscle fibre, a multinucleated cylindrical or polyhedral cell, 19 to 100 millimetres in diameter and 3 to 12 centimetres in length. A fine homogeneous hyaline sheath, the sarcolemma, surrounds the fibre and is applied exactly to its protoplasm. The protoplasm consists of a fundamental or nutrient granular, semi-liquid protoplasm on the surface of which are placed the nuclei, and a functional protoplasm, the fibrils of which are differentiated alternately and regularly throughout into clear and dark bands, giving rise to the striated appearance characteristic of voluntary muscle. The two ends of the fibre may be free, or may end in a tendon, or as is more usual, one end is free and the other ends in tendon.

The fibres are arranged into groups of 50-75 to form the primary bundles, being surrounded and supported by a framework of connective tissue. The primary bundles are similarly grouped into secondary bundles and in very large muscles the secondary are grouped into tertiary bundles. The supporting framework of connective tissue, the endomysium, also serves as a support for the blood vessels and nerves and springs from the connective tissue sheath which surrounds the muscle, the perimysium.

Blood Supply.

The blood supply of one muscle forms a vascular system which is independent of that of neighbouring muscles. The artery pierces the perimysium and breaks up into a network ramifying in the endomysium. Arriving at the primitive muscle bundle, the small arteries break up into arterioles which run in a direction perpendicular to the muscle fibres. From the arterioles come off long fine capillaries at right angles, two or three capillaries to each muscle fibre. These capillaries give off connecting branches which join up with those from the other capillaries of the same fibre. Thus the fibre is enclosed in a right-angled network of capillaries. From these capillaries arise the primitive venules which are perpendicular to the arterioles with which they alternate regularly. By the side of each arteriole is one small vein, running with each artery are two veins.

[1] Read at a Meeting of the Western Australian Branch of the British Medical Association on May 18, 1921.

Lymphatics.

Lymphatics only exist in the diaphragm and very large muscles, such as the *gluteus maximus* and *pectoralis major*, and in these they seem limited to the larger divisions of the endomysium.

Nerves.

The nerve supply consists of vasomotor, sensory and motor fibres. The motor nerve ending in a striated muscle fibre is known as the end-plate (Doyère, 1840).

About the middle of its length the muscle fibre presents a mass of granular protoplasm, thin in profile with a diameter equal to half the diameter of the fibre. This disc contains many large clear nuclei arranged around its periphery. The nerve fibre approaches the muscle fibre at right angles, the sheath of Henle blends with the sarcolemma, the sheath of Schwann ends indefinitely in the protoplasmic disc and the myelin sheath stops short of the muscle fibre.

The axis cylinder forms an arborization within the granular disc, the ends of the arborization being either attenuated or swollen. The physical state of a muscle depends on several factors. The amount of work demanded of it, the quality and quantity of blood reaching it, the rate of removal of its katobolic products and the character of the nervous impulses reaching it.

Atrophy of Muscle.

Atrophy of muscle may be divided into four main heads: simple, degenerative, neuropathic, primary myopathic or dystrophic. Simple atrophy is found in inanition from any cause (marantic atrophy). In many cases the condition is transient and the cause being removed, the muscle returns to normal. Should the process be long continued, degenerative changes are added. Atrophy from disuse may be produced by fracture of muscle, tendon or bone, ankylosis of joints, fixation by splints, or by prolonged voluntary inactivity. Excessive use will, after a period of compensatory hypertrophy, give rise to atrophy from exhaustion of nutrition.

In simple atrophy the changes are not very marked. The muscle is pale, soft and flabby, the fibres are thin and the transverse striation not as clear as normal.

Degenerative atrophies are those met with in intoxications, infections, chronic cachexias and anæmias and are due to changes in the quality of blood reaching the muscle. To these may be added those atrophies following atheroma, endarteritis, pressure of tumours on the main arteries, aneurysm and thrombosis of veins obstructing the free removal of katabolic products. The changes met with in the muscle fibres are vacuolation, fragmentation of the fibres, atrophy and finally disintegration of the fibre. The transverse striation is lost, the nuclei increase and giant cells are found in the fibre. The endomysium is increased relatively and often absolutely and in some cases there is a deposition of fat in the connective tissue.

In neuropathic atrophies the atrophy is confined to certain muscles, or groups of muscles, due to a lesion of the central or peripheral nervous system. In the former there is found a degeneration of the anterior horn cells of the spinal column, of the pyramidal tracts, the medulla or motor areas of the cerebral cortex. This results in what are known as progressive spinal, bulbar and cerebral atrophies. The extent of the atrophy resulting is dependent on the degree of disease present in the trophic centres and its localization governed by the particular centres involved. In certain diseases where there is a comparatively localized lesion, as in anterior poliomyelitis, certain forms of sclerosis, tumours and compression myelitis, the atrophy is confined to those small groups of muscles deriving their nutrition from the affected cells.

Some of the affections are progressive and lead to extensive muscle wasting and in some involving nearly all the principal muscles of the body.

Secondary muscular atrophy is also met with in certain affections of the peripheral nerves, such as multiple neuritis, chronic lead and arsenical poisoning and injuries to motor nerves by compression or division.

Somewhat closely allied to the progressive spinal atrophies is the so-called primary myopathy or progressive muscular dystrophy. This is characteristically a familial disease, so that it has been suggested that there is some congenital anomaly of development of the muscle. Others maintain that the primary disorder is in the central nervous system.

Clinicaly and anatomically there are two forms, the atrophic and the pseudo-hypertrophic. The changes in the muscle are similar to those described under degenerative atrophy, with the addition of a lipomatosis of the muscle in the pseudo-hypertrophic form.

Treatment in the neuropathic atrophies can only retard the progress. Massage and electricity have been found useful in lessening the discomfort in these cases.

In the examination of atrophied muscle or, more commonly, a group of atrophied muscles, we have to ascertain:

(*i.*) The cause of atrophy. This may entail a complete general and neurological examination.

(*ii.*) What muscles are involved.

(*iii.*) The condition of the atrophied muscles.

(*iv.*) Any alterations in cutaneous sensation accompanying the atrophy.

The cause may be obvious, as in drop foot from decubitis, wasted quadriceps following injury or disease of the knee joint, atrophy following anterior poliomyelitis. It may be obscure as in the neuropathic atrophies, the various forms of neuritis, cervical rib, etc..

(1) Does the atrophied group correspond to the distribution of a single nerve? Is the nerve lesion complete or partial?

(2) Does it correspond to a cord or root lesion?

(3) Does it correspond to a spinal segment?

(4) Does it correspond to a nuclear or supra-nuclear focus?

(5) Is there any bone, nerve or joint change?

(6) What is the condition of the skin? Is the dry scaliness of a complete nerve lesion or the bluish-red perspiring skin of a compressed nerve present?

(7) Does the blood supply of the affected limb compare with the sound one?

(8) Are there any factors aggravating the condition and retarding recovery? In this connexion I

may state that the rate of recovery of muscle and nerve in a chronic alcoholic is so slow that it is hardly worth while treating them.

The amount of wasting may be extreme and there may be no muscle substance left, the muscle tissue having undergone complete fibrous transformation, the blood supply being very poor and the perimysium having contracted right down. It is very difficult to tell in extreme cases without cutting down and taking a section of the muscle for examination. At the same time it is well to free the perimysium from the muscle.

Is voluntary movement present? In examining for this one should get complete relaxation. The weight of the limb should be borne by the physician, who should carry out the movement of the affected muscles asking the patient to assist him. There may only be a flicker of the tendon, still voluntary movement is present and this means that the muscle will recover. If voluntary movement be not obtained, the physician should ascertain if a faradic response is present. If it is present, the nature of the response, brisk or sluggish, the strength of current required to get a response compared with the strength required in a normal muscle should be noted. The rate at which the muscle tires to repeated stimuli should be compared with that of a healthy muscle. In *myasthenia gravis* the muscle soon tires to faradic stimuli, but not to galvanic. Sections of a myasthenic muscle demonstrated by Sir Frederick Mott showed degenerative changes in the end-plate. The speed at which they tire, seems to be due to the rapid using up of the exciting substance in the end plate. After a nerve has been divided faradic response sometimes remains for three weeks, the response gradually becoming feebler.

With a normal muscle and nerve the response to the negative closing current is greater than to the positive closing current. As degeneration of the nerve proceeds the response becomes equal and later the response to the positive closing current is greater than to the negative closing current. This is the so-called reaction of regeneration. In using this test it is necessary to register (*a*) the number of milliampères required to give the minimal response, (*b*) the highest rate of interruption of the metronome at which it is obtained, (*c*) the type of response (brisk or slow).

In the condenser response a curve is plotted of the amount of current in "microfarads" required to give a minimal contraction, the rate of interruption of the metronome per minute. A note is also made of the type of response.

It is sometimes necessary to give a probationary period of treatment by heat and massage before a reliable electro-diagnosis can be given.

Treatment.

This includes constitutional treatment and treatment of the cause, *e.g.*, suture of nerve, treatment of a joint injury, etc.. When the cause cannot be treated, as in some cases of nerve root injury or a gap in a nerve that cannot be spanned, treatment will consist where possible of substitution by tendon or nerve transplant, etc..

Rest is essential in the treatment of very wasted muscles. This means that the muscle fibres must under no circumstances be stretched. To obtain this the muscle is kept in a position nearer the position of full contraction than that of relaxation. A stretched muscle will not only not recover, but will undergo further degeneration. The position of rest may be maintained by splints, slings and other apparatus. A few examples are: the "cock-up" splint for wrist drop, the sling for the biceps, the abduction splint for the deltoid, the "calliper" for the quadriceps, the anterior spring for foot drop.

Some maintain that the atrophied muscle should not be stretched even for a second. I think this has been laid down to impress patients and attendants, the former that they must not leave the splint off while they go to a picnic and the latter that, while giving treatment, it is essential not to leave the muscles unsupported.

I think, however, that it is saving of time in the long run to put the joints through their full range once a day to prevent shortening of the muscle and the capsules of joints. It is necessary to give the antagonists some exercise, otherwise they may become very wasted and even functionally paralysed.

Massage is a valuable aid to treatment in cases of muscle atrophy. Its aims are: (1) to relieve pain and spasm by light rhythmical stroking, centripetal or centrifugal, whichever is the more comfortable. Its action is purely reflex. (2) To aid lymphatic and venous return by deep centripetal stroking. (3) To increase the arterial supply by causing a reflex dilatation of the arterioles by compression and vibration. The circulation of the affected part is thus increased and waste products are got rid of more quickly.

Massage has come in for a fair amount of adverse criticism and rightly so. I think the fault often lies with the doctor, who tells a patient to go and get massage and sends him to a half-trained or untrained masseur and leaves the form of treatment to a vigorous "rubber-down" or to one who has a little training and has formed original, if unscientific, ideas of what should be done. A trained masseur expects to receive instructions from the doctor as to disability, aim, time and what to avoid.

You can see the effect of a vigorous rubbing and compression of a scar which has just recently covered a septic compound fracture, or the effect of an energetic percussion and rubbing including passive movement of a lower limb in spasm after a compression of the sciatic nerve. The patient, already in great pain, is made to suffer torture, especially if the masseur is conscientious and gives a full hour's treatment.

Relaxation should precede massage of any form and for every purpose. The patient is placed in a comfortable position and the limb that is being treated, is well supported. Discomfort increases spasm. Passive movement implies movement by the masseur without the assistance or resistance, conscious or unconscious, of the patient. When you order passive movement you do not always intend the limb to be put through its full range of movement, but you do not always say so to the masseur.

The trained masseur will take it gradually, using pain as the sign to stop. The untrained masseur,

obedient, lacking in knowledge, but not in strength, may tear the newly formed granulation tissue and set up a reaction which will leave you a nice mass of scar tissue. You then say massage and passive movement are hopeless.

Re-education is very necessary in many cases of great atrophy. The patient finds that he cannot effectively use the wasted group of muscles and lays them aside, as it were, and forgets how to send the impulse from the cortex to them or, his nerve having recovered, the fails to realize that his once paralysed muscles are now only paresed. Re-education consists of getting the patient and the limb relaxed, doing the movement of the affected muscle and asking the patient to assist you. After a few movements you may see a flicker of the tendon. Show it to the patient and tell him that the muscle will now move at the order of his will. Make him do the movement three or four times and several times during the day, increasing the number of times on each successive day till the range of tendon movement is good. Then get him to sustain the contraction for one, two, three or four seconds each day till the muscle will move the limb for 10%, 20% or 30% of its range of action, the weight of the limb being supported through its ineffective range. Then follow exercises, gravity assisting, without assistance, with gradually increasing concentric resistance and lastly eccentric resistance.

Be sure before you begin re-education that the time is ripe for it, because it does look foolish to see anyone trying to re-educate a *tibialis anticus* with a 5 cm. gap in the external popliteal nerve.

Curative work is very valuable in restoring power to wasted muscles and of most value in getting back co-ordination which takes longer to recover than power. It also has an important effect on the morale of the patient.

Electricity, like massage, has come in for a fair amount of abuse. It has its uses and limitations. If properly applied by a competent person under instructions, it is useful; otherwise it is better left alone.

When a faradic response is present, graduated contraction will give more result in a month than massage will in three months. It is a useful adjunct to re-education.

When there is no faradic response, interrupted galvanism is used, purely to get a contraction of the muscle fibre to keep up its nutrition while the nerve is recovering.

A few contractions of each muscle every day is sufficient. If too many contractions of maximal amplitude are obtained, more harm than good is done.

The condenser is also used for the same purpose.

In the case of the Bristowe coil, it is not the electricity *per se* that does any good. It is simply a convenient means of stimulating the muscle to contract and of imitating the normal contraction and thus of giving the muscle exercise. The Bristowe coil gives the maximum muscle effect with the minimum skin effect.

In the case of galvanism I do not know whether the impulse acts on the remnants of the end plate or on the sarcoplasm. When the nerve is degenerated, the muscle replies with a slow sinuous contraction which seems to indicate that it is a protoplasm of a lower order than the fibrils that respond. I feel sure that interrupted galvanism is beneficial and retards the fibrous transformation that muscles deprived of their nerve supply undergo.

Radiant heat and whirlpool baths are useful precursors of massage and electricity. The heat increases the circulation and the resistance of the skin to the current is reduced. The whirlpool bath is also useful in relieving the spasm and pain of muscles and joints. Joints move more freely after the whirlpool treatment.

Special Cases.

(*i.*) The quadriceps in injuries about the knee joint. The atrophy of the quadriceps in these cases is more than can be accounted for by disuse. It is a reflex wasting. While the knee is resting the patient should be encouraged to contract his quadriceps a few times a day, provided it does not interfere with the condition for which the knee is being treated. Pain is the best indicator. As soon as possible the patient should be given massage to improve the circulation and reduce the œdema and graduated contractions with the Bristowe coil, special attention being given to the *vastus internus*.

Should the patient be allowed to get about with weakened ligaments and a wasted quadriceps, the knee is liable to give way and the patient will get a recurrence of the original process or a chronic recurrent synovitis. To prevent this a knee cage or "calliper" can be worn until the ligaments have shortened and the muscles developed. As the muscles become stronger, massage and Bristowe coil give way to graduated exercises. In the case of the knee cage it is important not to have the bands too wide, otherwise the compression will retard the recovery of the muscle.

(*ii.*) Drop foot from peripheral neuritis. While the patient is in bed the foot is maintained in a dorsi-flexed position by a bed boot and the weight of the bed clothes kept off the foot. When the pain has settled down, re-education of the muscles, Bristowe coil, massage and a spring to the boot in the day time to dorsi-flex the foor and a bed boot at night should be applied.

(*iii.*) Wrist drop from neuritis, crutch palsy, post-operative paralysis. A "cock-up" splint is required to keep the wrist dorsi-flexed. Massage, re-education, the Bristowe coil and exercises to the extensors of the wrist and fingers are needed.

(*iv.*) Facial paralysis, Bell's palsy. The paresed muscles are over-stretched by the pull of the unaffected muscles. This can be counteracted to a great extent by covering a piece of wire with narrow rubber tubing, bent into the shape of a hair pin and the curved portion bent to act as a cheek retractor. To the free ends is attached a cord to pass over the ear. Recovery is hastened by exercising the muscles in front of a looking glass.

Regeneration of Muscle.

Repair after injury or disease may be perfect or imperfect, according to the character and extent of the lesions. The rate of recovery depends on many factors, among which may be mentioned habits, loss of compensation by rapid recovery, neurasthenia

owing to rapid tiring of the centre for sending impulses to the motor cortex and psychasthenia owing to under-development of the centre which sends impulses to the motor cortex.

Histology of regeneration. Provided the nuclei and sarcolemma are not destroyed, the muscle fibre is capable of giving rise to new fibres. The nuclei divide by direct division or by mitosis, a multinucleated bud of protoplasm projects from the sarcolemma and grows out into the surrounding connective tissue or granulation tissue in the case of wounds and sepsis. At first the bud is not striated, but later it forms fibrils which become transversely striated. Multinucleated cells, sarcolytes, probably derived from free muscle corpuscles, are seen. These are phagocytic in action and may be seen to contain pigment, bits of necrotic muscle and detritus. They ultimately undergo necrosis and disappear.

Special splints and special forms of treatment are expensive and only within the reach of few. This should not be so and will not be so when a properly-equipped department is attached to every large public hospital. It has been amply proved in England that the cost of organized treatment plus loss in wages amounts to less than cost of "wait and see" treatment plus loss in wages.

BILHARZIOSIS IN WESTERN AUSTRALIA.

By **D. M. McWhae, C.M.G., C.B.E., M.D., M.R.C.P.,**
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and
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Resident Medical Officer, Repatriation General Hospital, Fremantle.

In August last year instructions were received from the Defence Department that all patients suffering from bilharziosis in Western Australia were to be collected in the Base Hospital and treated by intravenous injections of tartar emetic.

In all eleven patients have been sent by the State Quarantine Officer to the Hospital and have undergone treatment. All had been infected during 1916 by bathing or washing in the fresh water canal at Serapeum or Tel-el-Kebir. Some had also drunk water from this source.

The life history of the worm is briefly as follows: The *cercariæ* invade man by piercing his skin while bathing and it is possible that they may enter through the pharynx and œsophagus when the infected water is drunk. The *cercariæ* reach the liver, where they become mature and mate. They then migrate against the portal stream and deposit ova in the venules of certain viscera, chiefly the bladder and rectum. Dr. Hamilton Fairley describes how the ova are deposited in the tissue. The female worm works her way into veins of small diameter as far as she can go and then deposits an ovum with its spine directed posteriorly. She withdraws slightly, deposits another ovum and so on, until she finally withdraws, when the blood current resumes its ordinary course and drives the ovum before it; the ovum then pierces the vessel wall and passes into the perivascular tissue. It is then extruded through the mucous membrane by an inflammatory process which is dependent on an active toxic substance secreted by the *miracidium* contained in the ovum. Great numbers of ova may be thus deposited in the submucous tissue of the bladder and they lie separated from the cavity by a layer of granulation tissue which bleeds when the bladder becomes distended. Each ovum on reaching water hatches out a ciliated *miracidium* which has 48 hours in which to reach its intermediary host, certain species of fresh water snails of the genus *Bullinus*. The *miracidium* penetrates the liver of the snail and produces sporocysts. These produce *cercariæ*, which are discharged into water in enormous numbers for a period of weeks and thus are ready to invade man if he bathes or washes in this water.

There are two varieties of bilharzia, *viz.*, *Bilharzia hæmatobia*, producing terminal-spined ova, and *Bilharzia mansonii*, producing lateral-spined ova. The former are stated to be deposited in the bladder and the latter in the rectum, but in two of our patients lateral-spined ova have been found in the urine.

The symptoms of the eleven patients treated in this hospital may be divided into three groups:

(1) *Immediate Symptoms.*—These are an itching and burning of the skin immediately after bathing. Such symptoms occurred in only one patient.

(2) *General or Toxæmic Symptoms.*—These occured in five patients from three weeks to three months after the date of infection and consisted of urticaria, headache, general swelling, feeling of weakness and influenza-like symptoms.

(3) *Local Symptoms.*—(*a*) Bladder: Terminal hæmaturia was present in ten of the eleven patients and was first noticed from one to six months after the date of infection. It was often accompanied by a feeling of weakness in the back and pain on passing water. In three patients the hæmaturia has occurred daily without any cessation. In five patients periods of hæmaturia of several weeks' duration have alternated with periods of clear urine. In two patients hæmaturia had not been noticed for a year prior to entering the hospital. In one of these, however, blood was detected by microscopical examination.

(*b*) Rectum: In one patient the infection appeared to be limited to the rectum as hæmaturia or other bladder symptoms had not occurred. This patient has had so-called attacks of "dysentery" every six weeks, with bright red blood in the fæces. Three of the patients with bladder symptoms have also frequently had blood in the fæces due to rectal infection.

(*c*) Kidneys: One patient has had frequent attacks of renal colic on the left side and cystoscopic examination by Mr. Hadley showed that the left ureteric opening was inflamed and golf-hole shaped. Another patient has had "weakness in the back" and pain somewhat like renal colic. At the cystoscopic examination a group of small tubercles was seen about the left ureteric opening and blood was escaping from this orifice.

Miscellaneous symptoms are common, although slight in character. These are chiefly lumbar pain, slight abdominal pain, burning pain along the urethra during micturition, præcordial pain, palpitation, faintness, feeling of malaise and inability to do heavy

work. The patients as a whole, however, look well and feel fairly well.

Diagnosis.

This depends on finding the ova in the urine or fæces. The last 10 c.cm. of urine passed is centrifugalized and the characteristic ova with blood and pus cells are found under the microscope. Ova in fæces are very hard to find. As much fæces as will cover a shilling is shaken up in a test tube three-quarters full of water and is allowed to stand for 20 minutes. The supernatant cloudy fluid is then poured off and fresh water is added and shaken up as before. This is repeated until the supernatant fluid is clear. The residue is then examined with the microscope.

Ova, blood cells and pus cells have been found in all the patients with hæmaturia excepting one and we have no doubt that ova will be found in this patient when he is re-admitted to hospital.

One doubtful ovum was found in the mucus of the fæces of the patient with rectal infection. The presence of eosinophilia and of lesions detected on cystoscopic examination offer confirmatory evidence of bilharziosis.

Infectivity.

There seems little likelihood of spontaneous cure. For over four years eight of the patients with hæmaturia have continued to pass each day many ova. Of the remaining three patients one has left the State, one is again required in hospital for further examination and one—the patient with rectal infection—appears to be cured.

Fresh water, snails and warm weather—ideal conditions for the spread of bilharzia—occur in this country in abundance. Dr. W. H. Nelson reported in the *Australasian Medical Gazette* of November 9, 1912, three or four cases of bilharziosis in which the infection was traced to an ex-soldier from the South African war. Effective treatment of these patients is therefore of great importance to the Commonwealth. Our observations on treatment by tartar emetic are therefore submitted.

Treatment.

Technique.

A solution of tartar emetic containing 0.03 gramme in 10 c.cm. of normal saline solution is prepared. Intravenous injections are given on alternate days, *viz.*, a first injection of 0.03 grm., a second of 0.06 grm., a third of 0.09 grm., a fourth of 0.12 grm. and subsequently all injections are of 0.12 grm..

No untoward symptoms of any kind have occurred as a result of the injections. Frequently these cause a sharp rise of temperature and very occasionally a slight rigor or some vomiting, but the disturbance is not serious and is soon over.

Criteria of Cure.

Christopherson states that 1.5 to 1.8 grammes administered in this way invariably cures, although dead ova may be passed for years.

F. O. Lasbrey and R. B. Coleman in the *British Medical Journal* of February 26, 1921, in a report on the treatment of 1,000 cases of bilharziosis state that they stopped the injections when all ova were found dead on three consecutive days. They had difficulty in following up their cases, but report that they have never seen living eggs re-appear after the completion of a successful course and that there has been a complete relief from all symptoms in several patients seen by them some months after treatment. The amount of tartar emetic injected by them

Case.	First Treatment. (Amount of tartar emetic.)	Time between termination of treatment and re-examination.	Effect of first treatment on symptoms.	Effect of first treatment as determined by examination of urine on re-admission. (Urine examined daily during period of re-admission to hospital.)
1	1.86 grammes	5 months	Gained 9 kilograms; hæmaturia not noticed by patient since treatment	Live ova still present with blood and pus; ova hatch
2	1.62 grammes	6 months	Gained 6.35 kilograms, but states that hæmaturia returned 14 days after treatment and has continued as bad as ever	Live ova still present with blood and pus; ova hatch
3	1.53 grammes	6 months	Symptoms have disappeared since treatment	Live ova still present with much blood and pus; ova hatch
4	1.53 grammes	4 months	Weight stationary; hæmaturia appeared 14 days before re-admission to hospital; occasional palpitation and præcordial pains	Live ova still present with much blood and pus; ova hatch
5	1.034 grammes	6 months	Gained 450 grammes; during 4 months only noticed hæmaturia once, but for past two months passing "blood, lumps and pus" as bad as ever	Live ova still present with much blood and pus; ova hatch
6	1.38 grammes	4 months	Lost 5 kilograms; hæmaturia and all symptoms relieved for 2½ months; in past 1½ months they have returned	Live ova still present with blood and pus; ova hatch

varied from 0.72 to 1.62 grammes. These statements are not in accord with our own observations. Our patients have been given a course of treatment with instructions to report for re-examination in three months after the date of discharge. So far only seven patients have reported. One of these was suffering from rectal infection only and is therefore excluded from the following table, which shows for the six patients the amount of tartar emetic given with its resulting effect on symptoms and urine.

One patient was given only 1.035 grms. because he had to leave the hospital for business reasons. The remainder were given from 1.38 to 1.86 grms. and treatment was stopped when live ova were not found in the urine.

It was thus apparent that one course of treatment was insufficient to cure these patients. A further course of treatment was therefore given and its effect on the ova were carefully watched each day. This is described in the following table:

Case.	Second Treatment. (Amount of tartar emetic given to April 1, 1921.)	Effect on Urine. (Urine examined daily.)	Hatching at end of treatment (April 27 to May 1)
1	0.9 gramme	Live ova decreased markedly and from April 27 to May 1 only an occasional degenerated ova found; blood and pus decreased steadily	Ova do not hatch
2	1.26 grammes	Live ova decreased markedly and from April 27 to May 1 33% of all ova are degenerated; blood and pus decreased	Ova do not hatch
3	1.5 grammes	Live ova decreased markedly and from April 27 to May 1 only one degenerated ovum found; blood and pus markedly decreased	Ova do not hatch
4	1.5 grammes	Live ova decreased markedly and from April 27 to May 1 33% of all ova are degenerated; blood and pus still present in fairly large amount	Several ova hatched on May 1
5	1.62 grammes	Live ova decreased markedly and during last four days of treatment all ova dead; blood and pus very slight in amount	Ova do not hatch
6	1.74 grammes	Live ova disappeared by end of first week of treatment; for remaining month of treatment no live ova found and during this period only an occasional blood and pus cell seen; no ova present	No ova found for last fifteen days of treatment

Thus in these cases the tartar emetic has had a specific destructive effect on bilharzia ova and it has been noticed that during the early stages of treatment areas of bladder mucosa containing large numbers of ova may be exfoliated.

The position with regard to the remaining five patients who have not yet been re-admitted to hospital is shown in the following table.

Case.	First Treatment. (Amount of tartar emetic given.)	Remarks.
7	1.53 grammes	Not yet returned for re-examination; mixed bladder and rectal infection; small ulcer revealed by sigmoidoscope 3.75 cm. up rectum
8	1.62 grammes	Not yet returned for re-examination
9	1.35 grammes	Not yet returned for re-examination
10	0.96 gramme	Was suffering from rectal bilharziosis; only one doubtful ovum seen in mucus of fæces; no blood in fæces for many months; appears to be cured
11	0.9 gramme	Left hospital and State before completing treatment

The following conclusions therefore seem justified:

(1) One course of treatment with tartar emetic is insufficient to destroy all bilharzia ova permanently, despite the following two facts:

(*a*) That at the termination of treatment ova may not be found in the urine; or

(*b*) That all the ova found therein at the termination of treatment may be dead.

(2) Tartar emetic has a specified destructive action on the bilharzia ovum and, therefore, probably also on the adult worms.

(3) Although one course of treatment is insufficient to cure, it is extremely probable that two or more courses will in the end completely stamp out the disease.

Note by Dr. D. M. McWhae.

Except for a comparatively small number of examinations of urine and fæces carried out by the Public Health Department in the early stages of our work, all examinations of urine and fæces, together with all the injections, have been performed by Dr. Jaggar and the painstaking and careful work of this officer has alone rendered the investigation possible.

It is proposed to recall patients for re-examination three months after the termination of their second course of treatment. This report is required by the Quarantine Department in Melbourne; hence its preparation before the completion of our investigation.

RADICAL EXTIRPATION OF CALCIFIED HYDATID CYSTS WITH ADVENTITIOUS CAPSULE COMPLETE.[1]

By **C. E. Corlette, M.D., Ch.M. (Syd.),**
Honorary Surgeon to the Sydney Hospital.

In operating for hydatid disease of the liver, one occasionally meets with cases that present a hard, calcified adventitious capsule. The cysts which I have seen in this condition have been small to moderate in size, never extremely large. Such hydatids offer some problems to the surgeon. The difficulty that arises is created by the hardness of the calcified wall within which the parasite cyst is enclosed. If the cyst is treated by opening, removal of the parasite, marsupialization and drainage, a permanent non-collapsible cavity might remain. Another plan is to refrain from interfering and to close up the wound. This is far better surgery than opening and marsupialization and illustrates the fact that the least operating is sometimes the best surgery. Another plan is to remove the parasitic cyst in the ordinary way and then to cut away with scissors as much of the capsule as may project beyond the liver, leaving the intrahepatic portion; the liver is then dropped back. I have found, however, that there is another method possible in some cases.

In February, 1919, I had to deal with a hydatid about as large as a cocoanut growing in and on the left lobe of the liver. When this was incised, its wall was found to be calcified. It contained a large quantity of thick, dark, hydatid material, which was removed by a spoon. The cavity was then further cleaned out by the usual methods. It was at first intended to remove only the free portion of the adventitia, but, after catching it up with forceps, instead of clipping it off, I tried to see whether it was intimately connected with the proper tissue of the liver. I found that, with care and by using a swab-stick for dissection, I could separate it from the under surface of the liver. Still proceeding carefully, I gradually separated the calcified shell, not only from the liver, but from the gastro-hepatic omentum, anterior wall of stomach and pylorus. No vessels were encountered entering the capsule, but large vessels were exposed adjacent to it. Bleeding was not troublesome, though some vessels required ligation. The operation amounted to a radical removal of the hydatid and its adventitious capsule. The abdomen was closed and recovery was uneventful.

More recently, in May, 1921, I had to deal with a hydatid about the size of an orange, growing from the right lobe of the liver, partly embedded and partly projecting. Finding it to be hard, I made use of my previous experience, just related. I did not attempt to open the cyst, but tried to see whether it had any close vascular connexion with the liver substance. I found that in this case also the calcified adventitious capsule had become practically the same as a dead foreign body and it could be separated like an embedded stone. With extremely careful gauze dissection, it was gradually freed and removed unopened, no more hæmorrhage being encountered than one meets with in an ordinary cholecystectomy. The wound was closed up and the patient recovered uneventfully, leaving the hospital in seventeen days.

These two examples show that in certain cases of calcified hydatid a radical extirpation, including the capsule, is a safe operation and one seriously to be considered when such cases are met with. Of course, every surgeon undertaking such work must have sufficient judgement and experience to know what he may do or may not do with safety and he should know when to desist. I suppose it is necessary also, to add the warning that it would be the worst possible surgery to attempt an operation of this kind for any other type of cyst. Such operations have indeed been done and recorded by surgeons, or let us rather say, by operators, but they have invariably killed their patients. Perhaps I should not say "invariably," for I have found one instance of survival recorded.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on June 10, 1921.

Reports of Cases.

A PLASTIC OPERATION ON THE FACE.[1]

By **Howard Bullock, M.B., Ch.M. (Syd.), F.R.C.S. (Eng.), B.Sc. (Oxon.),**
Honorary Assistant Surgeon, Sydney Hospital.

The case of C.R., a woman of 56 years, is of interest in that the whole of the lower lip, as shown in Figure I., was destroyed by a squamous-celled carcinoma. Dr. P. E. Walton Smith made the pathological diagnosis. The growth had also invaded portion of the chin and the outer table of the mandible to the right of and adjoining the *symphysis menti*.

FIGURE I..
Showing growth and incisions used.

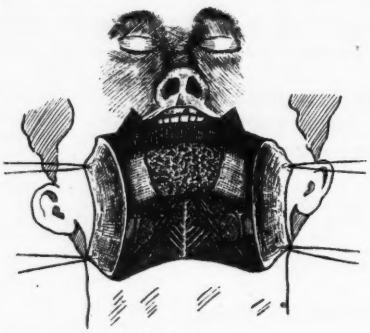

FIGURE II..
Showing the skin-flaps dissected back, the portion of the mandible removed, muscles and glands of the sub-mental triangle and portion of the muscle and glands of the sub-maxillary triangles.

FIGURE III..
Showing skin-flaps in position and drainage tube.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on June 10, 1921.

While attending the clinics at Berlin in 1909 and 1912, at Edinburgh in 1911 and also for several years in London, I observed that, when there existed any doubt as to whether the malignant disease had involved the bone, the bone was sacrificed freely and wide of the disease. The same routine has been invariably carried out in the hospitals of Sydney. It had always been my practice to work on similar lines until

FIGURE IV..
Growth removed with V-shaped portion of lip taken out for microscopical examination. A—A^1, B—B^1 shows red margin of apparently healthy lip. A—B^1 angle of the growth.

I operated upon this patient. The outer table of the mandible, with the diploë, about 3 cm. wide of the seat of the cancer, was removed from each side. The inner table was scraped and thinned to a mere shell and was then well cauterized with the actual cautery.

A quadrilateral area (Figure I., A^1BB^1D) was cut *en masse* down to the bone and removed. The mandible was treated as above described. Starting from the angles of the mouth (A and D), I made incisions AYC and DY^1C^1 through the whole thickness of the cheek, taking care not to injure the ducts of the parotid salivary glands. Triangular-shaped incisions AXY and DX^1Y^1 were then made through the skin only, the mucous membrane being left behind and used in the construction of a red margin for the

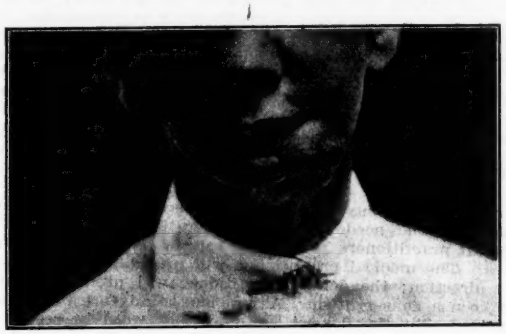

FIGURE V..
Photograph taken on June 6, 1921, after the operation.

angles of the mouth. These incisions were used to remove the puckering of the skin above and to the outer side of the angles of the mouth. Incisions BE and B^1E^1 were then made below the chin along the folds of the neck to the outer margins of the sterno-mastoid muscles. The skin of the flaps A^1BE and $D^1B^1E^1$ were dissected up from the platysma, well over the mandible. The skin along the line EBB^1E^1 was similarly treated. The platysma and contents of the sub-maxillary and sub-mental triangles were then thoroughly cleared away and after ligation of the external maxillary arteries and veins, the supra-mandibular glands were carefully removed. The upper deep cervical glands were likewise removed on both sides. The flaps were then brought together over the well cauterized bone and a new lip fashioned. Drainage tubes, as shown in Figure III., were left in for 24 hours.

The patient had an uninterrupted recovery. The operation was performed in February, 1918, and when the patient was shown at the clinical meeting of the New South Wales Branch of the British Medical Association held at Sydney Hospital on June 10, 1921, three years and three months after operation, there were no signs of recurrence. It is of interest to record that a total hysterectomy for multiple uterine fibroids was carried out by me in 1919.

The unusual features about this case are that the mandible was obviously eaten into by the growth and that, though the bone was not subjected to the radical procedure of a complete sectional removal, no recurrence has so far taken place.

DISARTICULATION AT THE HIP-JOINT FOR SARCOMA OF THE FEMUR.[1]

By **T. Fiaschi, M.D., M.Ch. (Univ. Pisa),**
Honorary Consulting Surgeon, Sydney Hospital.

The patient with whom we are concerned, a young man of 22 years and a resident of Mudgee, was first seen by me in consultation with Dr. R. Steer Bowker. We found him to be suffering from a large tumour in the upper third of the femur.

A small incision for diagnostic purposes had already been made and a small specimen removed. It had been pronounced by a pathologist to be a sarcoma. Other surgeons had seen the patient and pronounced the condition inoperable. However, we thought it right to give the young man a chance. He was slightly feverish at the time and a few lymphatic glands in the groin were somewhat enlarged, but we accounted for this by some irritation, the result of the exploratory incision in the sarcoma.

The operation took place on November 14, 1919. To avoid the greatest danger of this formidable operation, *viz.*, hæmorrhage, we applied Momburg's aortic tourniquet. Momburg's method consists in the application round the body at the level of the umbilicus of a stout india-rubber tube about 2 cm. in diameter, tightened sufficiently to stop all pulsation in the femoral arteries. Before doing this we had applied a tourniquet around the upper third of the healthy thigh and another around the affected thigh, just above the tumour, to prevent sudden lowering of the blood pressure after release of the aortic tourniquet. Ether was administered by Dr. P. Fiaschi by the intra-tracheal method and attention was paid by him to the blood pressure.

I was kindly assisted by Dr. R. S. Bowker and we performed Farabœuf's disarticulation at the hip-joint by the anterior racket method. All proceeded very well; there was no hæmorrhage to worry us and we were able to ligate all the blood vessels leisurely, as we worked.

Before dividing the sciatic nerve, I injected into it some Billot's stovaine to prevent shock. Before bringing together the flaps, I removed as many lymphatic glands from the groin as I could. The flaps came together very well in a longitudinal line.

The reading of the systolic blood pressure before operation was 125 mm. of mercury. On disarticulation the reading was 90 mm.. On removal of Momburg's tourniquet it was 110 mm.. On removal of the tourniquet from the left leg it was 120 mm..

The patient made a good recovery. The stump was considerably swollen for the first four days, but the swelling gradually subsided. The temperature on the second day rose to 40.2° C. and the pulse rate was 156; but under the administration of quinine in 0.3 gramme doses and with the aid of two injections of surgical pituitrin, he soon recovered a normal pulse and temperature.

The specimen was examined by Professor D. A. Welsh,

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on June 10, 1921.

and found to be a sarcoma of the large spindle and mixed-cell variety, such as is common in bone and periosteum.

To make sure against the risk of recurrence of the sarcoma, my son, Dr P. Fiaschi, began on December 12, 1919, to administer to the patient injections of Coley's serum, beginning with a dose of 0.017 c.cm. and gradually increasing to 0.2 c.cm.. Nine injections in all were given and were followed by reactions characterized by a temperature which rose as high as 39.4° C.. On his return to Mudgee these were continued for two months by Dr. F. Challands and given every three days. The patient has since enjoyed good health. Fortunately, so far, nearly 18 months from operation, no sign of return of the sarcoma has taken place.

Remarks.

My objects in demonstrating this case are: (1) To combat surgical pessimism on the question of amputation for sarcoma of long bones. Certainly the statistics in regard to recurrence of the disease are very unfavourable, but I think that patients should be given a chance; and amputation followed by treatment with Coley's serum is far better than leaving them to their inevitable fatal ending.

(2) To demonstrate once more the effectiveness and simplicity of Momburg's method of hæmostasis. The immediate operative mortality for disarticulation at the hip-joint is 40%. This mortality has been chiefly due to hæmorrhage. It will be curious to see now how far this mortality will be reduced by the adoption of Momburg's hæmostasis.

In addition to this case. there have been two other cases in which I have performed under Momburg's hæmostasis Jaboulay's inter-ilio-abdominal amputation. In the first case the patient was a woman suffering from advanced tuberculosis of the left *os innominatum*. She was operated upon by me in the Sydney Hospital. She stood the operation well. There was very little hæmorrhage and the surgical recovery was good, but she died later on of the original disease. In the other case the patient was an Italian soldier operated upon by me in the 73rd Italian war hospital at Schio. This man had been severely wounded in the upper part of the left thigh and pelvis by numerous large fragments of shell and irregular resection of the hip-joint had been done a few weeks before, but extensive suppuration and sepsis had followed, reducing the man's strength to the lowest ebb. I performed Jaboulay's inter-ilio-abdominal amputation under Momburg's hæmostasis. In this case also the hæmorrhage was surprisingly small. He made a good recovery and one year ago I got a post card from him, saying that he was keeping well.

Before the war, in a paper read before the Military Section of the Australasian Medical Congress held at Sydney in 1911, I advocated Momburg's method as likely to be of great help to the military surgeon. This opinion has, I believe, been justified.

Reviews.

PYORRHŒA.

Dentists are in the habit of claiming that the medical practitioner is treading on unsafe ground when he poaches on the oral preserves. In these circumstances a book on *pyorrhœa alveolaris*, written by a throat surgeon, might meet with little welcome from the dental profession. Mr. Crow's book, however, will probably gain popularity with both dental and medical practitioners, for it carries as a preface a whole-hearted recommendation from Sir Frank Colyer.[1] The author is frank from the first sentence of his book. He refers to "the overwhelming importance of *pyorrhœa alveolaris* in the causation of many diseases and minor complaints." Sir Frank Colyer, in the foreword, makes the bold assertion that the swallowing of the septic material leads indirectly to toxic conditions of the rest of the alimentary canal and that toxins pass into the general circulation by way of the medullary tissues. Both endeavour to evade criticism by admitting that it is difficult to establish a causal connexion between the disease and remote disturbances. It is possible that the general view that pyorrhœa leads to systemic infections may be correct. The evidence is of the slenderest character; it is based exclusively on the occasional disappearance of general symptoms after the effective treatment of the local dental affection. No one has demonstrated an intestinal lesion, resulting from the swallowing of pus from a tooth pocket. The postulation of a hypothetical toxin passing from a tooth into the blood stream is unconvincing, since it is at present impossible to identify a poisonous substance in the blood that has its source around an infected tooth. Loose statements of this kind are unscientific and do not enhance the value of any book. Unfortunately, they are repeated, parrot-fashion, by the vast majority of writers on pyorrhœa and other local diseases.

Mr. Crow deals with pyorrhœa from the surgeon's point of view. He traces the disease from its earliest beginnings, when the lesion is limited to the gingival margin. He is unconcerned with the biological and chemical processes and focuses his attention on the physical problem as it appears to the surgeon's eye. An infection, characterized by pus formation, starts at a minute point. Mr. Crow measures the surface which may be affected if the pathological process is not arrested and endeavours to persuade his readers that the danger to the patient is proportionate to the extent of area involved. In connexion with the diagnosis he describes the macroscopical and skiagraphical appearances which are familiar to every dentist and to the majority of medical practitioners. In many instances the determination of the presence or absence of pyorrhœa rests with the X-ray appearances. The author neglects to deal with the more difficult forms of differential diagnosis, nor does he state on what signs an early infection of the lingual margin of the mucosa is to be based. His sovereign remedy for undoubted pyorrhœa is extraction and consequently it is scarcely surprising to find a description of the extracted pyorrhœal tooth among the clinical signs.

Perhaps the most interesting part of the book is that in which Mr. Crow attributes appendicular abscess, duodenal or gastric ulcer, *otitis media*, cholecystitis, pyæmia, pernicious anæmia, malignant endocarditis, pneumonia, chronic nephritis, Ludwig's angina and many other conditions to pyorrhœa. It must be noted that this discussion is introduced in a chapter headed "Symptoms"! He argues without evidence and without proof. Many of his observations are interesting, many are important and all bear the signs of an enthusiast. His deductions are hazards in the dark; but they may reach near the truth. Bacteriology should be able to help to a slight extent; but if the presence of a suppurative lesion, starting in the gingival trough, spreading to the alveolus and causing destruction of the bone and eventually of the roots, is followed by a disturbance in a remote part of the body, the determination of the causal organism of the primary process need not provide information concerning the pathogenesis of the secondary involvement. If a soluble toxin is produced, it is conceivable that it might exert some damaging influence in other tissues. To prove this, the bio-chemist, and not the bacteriologist, would be needed. Mr. Crow expresses the opinion that, until the bacteriology of pyorrhœa has become less confused, only one overwhelming proof is available for his contention. His "overwhelming proof" is the effect of the removal of the suspected source of trouble. Would not the health of the patient improve if two unconnected lesions co-existed and one of them were removed? Would not benefit accrue if purely local lesions in the mouth were healed. The book illustrates the need for the collaboration of dentists with medical practitioners in the interests of patients with pyorrhœa. The medical practitioner will probably go too far in one direction; the dental practitioner will probably exhibit too keen a conservatism. A judicious admixture of opinion, provided that the dentist has the final word in regard to extraction, should be safe and satisfactory. Mr. Crow's book is certainly worth buying and reading carefully.

[1] Pyorrhœa Alveolaris in its Clinical Aspect, by D. A. Crow, M.B., Ch.B., with a Foreword by Sir Frank Colyer, F.R.C.S., L.D.S.; 1921. London: Baillière, Tindall & Cox; Crown 8vo., pp. 111. Price, 6s. net.

THE WAR MEMORIAL FUND IN VICTORIA.

The following have contributed to the War Memorial Fund of the Victorian Branch of the British Medical Association:

Agnew, J. F.; Black, J. P. M.; Blackburn, John H.; Cowen, H. O.; Gutteridge, E. W.; Johnston, W. W. S.; Laurie, W. S.; Mackay, J. S.; Noyes, A. W. F.; Potter, W. L.; Sutherland, B. M.; Wilson, A. M..

The Medical Journal of Australia.

SATURDAY, SEPTEMBER 17, 1921.

The Incorporation of Branches.

A day or two after the Federal Committee of the British Medical Association in Australia had completed its last session, a cable was received from Dr. R. H. Todd, the plenipotentiary of the Branches, announcing that the Representative Body had conceded all the points for which he had asked in the name of the Branches in Australia. The fact was announced in our issue of July 30, 1921, and some general statements concerning the significance of the altered conditions were appended. The *Supplement to the British Medical Journal* of July 23, 1921, containing a report of the debate in the Representative Meeting on July 16, 1921, has now come to hand. It should be studied with care and studious attention by every member of the British Medical Association. In order that the procedure may be followed, it is necessary to bear in mind the details of the constitution of the Association and also to refer to the reports of the Council in the *Supplements to the British Medical Journal* of March 5 and April 30, 1921. The key note of the situation is contained in one sentence of the speech of Dr. Russell Coombe, the Chairman of the Organization Committee: "He (the Solicitor of the Association) had come to realize that federation was not what the overseas bodies wanted." It is unnecessary at this stage of the negotiations to dwell on the fact that the Federal Committee had made this abundantly evident in the many communications addressed to the Council of the Association during the past two years or more. It would appear that Mr. Hempson, the Solicitor, and the members of the Organization Committee, having realized that the Branches in Australia understood exactly what they could do under the old regime and what they wished to do to carry out the objects of the Association, discussed the problem from the premises set up by Mr. Gore-Browne and others acting as counsel for the Association. The Organization Committee was not prepared to challenge the soundness of the older legal opinions. Dr. Todd was unable to persuade the members to adopt the attitude that it was legally possible for a Branch of the Association to become incorporated and at the same time to retain its status within the Associaion. He therefore chose the wise course of discussing the legal point with Mr. Colquhoun Dill and Mr. Hempson. Mr. Dill listened to Dr. Todd's arguments and agreed with him that there was no inherent obstacle in the constitution, as set out in the Memorandum of Association, to the adoption of the proposals of the Federal Committee. A written opinion was drawn up by Mr. Colquhoun Dill and this document was presented at the conference of July 5, 1921. Mr. Russell Coombe holds that Mr. Dill's opinion created an entirely new situation. This new situation has been discussed in Australia and in this *Journal* on many occasions during the past two years. The conference had been called to consider certain proposals put forward by the Council for the purpose of enabling the Association "to become in part a federation." The members had to be won over to the changed situation. Dr. Todd had the task of removing from the minds of the members the unsatisfactory proposal that a Branch, if it became incorporated under the statutes of a colony or State, would have to sever its connexion with the British Medical Association and then beg to be received as an affiliated body, a body with restricted rights, a body loosely linked to the parent Association through newly created machinery. He had the task of asking them to recommend to the Representative Body that the draft articles and by-laws contained in the reports of the Council be withdrawn and that fresh articles and by-laws be drawn up, whereby the procedure might be regularized for an oversea Branch incorporated under the *Companies Acts*. It may be assumed that the difficulties which Dr. Todd overcame during the conference, were by no mans small. It was the second stage in his campaign of education and events prove that it was well performed. The third stage was probably less formidable. Mr. Russell Coombe, in the name of his Committee, asked the Representative Body to withdraw the proposals of the Council, to which reference has been made above.

In the next place he asked them to accept the following proposal:

> That, if possible, provision be made in regulations of the Association to allow the overseas Branches to retain the character and status, the rights, powers and duties of Branches, although incorporated, such steps being taken as may be necessary to protect the Association from liability for any act of the incorporated body.

Dr. Todd was then asked to explain to the meeting the views and arguments in favour of the motion before the meeting. His splendid address must have convinced the representatives. Had there been any doubt left in the minds of his listeners at the end of his speech, this would certainly have been dispelled by a trump card, held in reserve. In reply to a question by Dr. J. A. Macdonald, he quoted a further report from Mr. Colquhoun Dill, dated July 11, 1921, to the effect that in his opinion the Memorandum of Association was not inconsistent with the admission of corporate bodies as Branches of the Association. The motion was carried with acclamation. The Representative Body has thus scrapped the federation proposals of the Council, and has instructed the Council to submit to the Representative Meeting in 1922 draft articles and by-laws to enable oversea Branches to secure by incorporation all the rights, powers and duties of the parent Association, without sacrificing their status as Branches. The delay is of small moment, in view of the importance of the ultimate changes. It is a little unfortunate that the anomalous words "if possible" were not removed, for it has been shown that the proposal is eminently practicable and legally possible.

The last resolution of the Representative Body in connexion with the oversea Branches is an instruction to the Council to submit regulations for the establishment of Federal Councils to carry out on behalf of the local Branches in the Dominions and India the objects of the Association and generally to have the powers and duties of the existing Federal Committees. It had been anticipated in these columns that the Federal Councils would have the right of incorporation. From the very brief record of the proceedings, it would seem as if this proposal had not been contemplated. In practice, however, they would not be enabled to carry out all the objects of the Association, as set out in the Memorandum of Association, unless they were incorporated in some manner. No doubt this point will be settled when the provisions suggested by the Council are submitted to the Representative Body next year.

The Branches of the British Medical Association in Australia may look forward with pleasant anticipation to a future of extended and increased utility.

THE SURGERY OF THE PITUITARY GLAND.

Hidden in the *sella turcica* of the sphenoid bone, at the base of the brain, is a ductless gland of obscure proportions, called the pituitary gland. It measures less than 1.3 cm. in its widest dimension and weighs less than three-quarters of a gramme. Yet this small, unobstrusive structure has an enormous significance in the regulation of some of the most important processes of the body. So significent is its influence that a surgeon has been tempted to leave for a time the strict path of his calling and wander into the wood of metaphysical and moral speculation. If, like the babes of the fable, he becomes lost in this strange wood, unlike them he emerges once again on to the safe road of his own sphere. Dr. A. J. Walton[1] is shocked to realize "how largely our appearances, our actions, our morals and our very destinies are dependent upon our internal secretions over which we have no control." Is a man, he asks, who has a lowered pituitary output to to be regarded as a mental weakling and a dullard, while he who is blessed with a more efficient secretion, is acclaimed for his intellectual brilliance? Is morality to be measured in terms of sexual glandular activity and artistic temperament by the efficiency of the thyreoid? And, having strayed into the dangerous wood of moral philosophy, he draws discreetly back with the hope that a careful study of the internal secretions will one day allow us to give to those whom Nature has denied, the two boons of life—physical fitness and "sunshine in the open country."

The functions of the pituitary gland have been investigated by many and, while the present state of our knowledge is far from adequate, much of the physiology of this curious body has been elucidated. We know, for example, that the anterior lobe influences growth and development. Its secretion has a certain relationship to calcium metabolism and to the growth of bone, cartilage and connective tissue. The secretions of the posterior lobe and the *pars intermedia* influence the metabolism of the carbo-hydrates and possibly of the fats. Hyperpituitarism is associated with diminished carbo-hydrate tolerance and sometimes with glycosuria. In hypopituitarism this tolerance is increased, so that in Fröhlich's disease and in Dercum's disease (*adiposis dolorosa*) the patients are invariably fat. The posterior lobe also secretes a substance which raises the blood pressure, another which stimulates diuresis, a third which acts as a galactogue and a fourth which causes powerful contraction of unstriped muscle. How far these substances are identical or individual it is as yet impossible to tell. We know of no definite disease associated with an increase of function of the posterior

[1] *The Lancet*, June 4, 1921.

lobe, though acromegaly is not a morbid condition of the anterior lobe alone. Deficiency of posterior lobe secretion leads to the extraordinary morbid states known as *dystrophia adiposo-genitalis* (Fröhlich) and *adiposis dolorosa*. The former is a disease of the young, the latter an affection of adults. In both there is a large over-growth of subcutaneous fat, which is the adult condition is deposited in large, irregular, tender masses. A male child who exhibits Fröhlich's syndrome, has a body which assumes the characteristics of the female figure. The buttocks and thighs enlarge, the breasts became prominent, the skin soft, the voice high-pitched. The genitals remain small and undeveloped and there is no growth of axillary or pubic hair. The blood pressure is low and the tolerance to carbo-hydrates is increased. The assumption that this disease is due to an involvement of the pituitary gland is justified by skiagraphic appearances, which show enlargement of the *sella turcica* and by the finding of a tumour or cyst at operation or autopsy. Dr. Walton describes the case of a girl of 14 years who manifested the disease in a typical form. At operation a cyst lying in the pituitary fossa was punctured and drained. The impeachment of the gland as the cause of the other condition, *adiposis dolorosa*, rests, however, on little pathological evidence. But the analogous nature of the condition to Fröhlich's disease and the correspondence of many of its features to the known physiological effects of the hypophysis justifies the assumption that that gland is at fault.

Not less interesting are disturbances of function of the anterior lobe. The hyperpituitarism which leads to the disease called acromegaly, is familiar to every physician. Acromegaly is not a disease exclusively of the anterior lobe, but disturbance of the anterior lobe is responsible for its main clinical features. The growth in height, the general hypertrophy of the face, the downward enlargement of the lower jaw, the big, half-lolling tongue, the enormous ears, the spade-like broadening of the hands and feet and the osseous outgrowths involving the joints and skeleton form a picture which is vivid in its Gargantuan realism. In women amenorrhœa is an early symptom and the breasts become small. The external genitals, however, become hypertrophied. The voice is deep and the hair becomes coarse. The unfortunate woman who is the victim of this disease, assumes more and more the general build and appearance of the male body. Polyuria and glycosuria are not uncommon. As in every disease of the pituitary gland bitemporal hemaniopia, due to pressure of a cyst or tumour on the decussating fibres of the optic chiasma, may be a striking feature. Optic atrophy, partial or complete, may eclipse "the talent which is death to hide." As in the tabetic, the fear of blindness hangs over the acromegalic like a pall. That the intelligence remains good serves only to increase the patient's realization of the change which has come upon him. Acromegaly occurs in adult life. Brissard showed many years ago that the gigantism of the young was closely related to acromegaly. One out of every five acromegalics is a giant and two of every five giants develope acromegaly. The opposite condition to gigantism is ateleiosis, more commonly known as Lorain's disease. In this disease the child fails to grow with age. His body is uniformly dwarfed, unlike the condition in achondroplasia, in which the extremities chiefly suffer. The skin is soft as a woman's, the voice shrill, the face and body hairless. Possibly not a few of the quaint citizens of "Tiny Town" belonged to this group. In ateleiosis the skiagraphic appearances of the *sella turcica* are very similar to those of other pituitary conditions.

Dr. Walton has described the surgical treatment of pituitary diseases. The method of approach which he favours, is the orbito-frontal route, initiated by Krause and perfected by C. H. Frazier. An opening is effected into the cranium by turning down a flap of frontal bone, the *dura mater* is raised from the orbital roof and then incised till the optic nerve and chiasma become completely visible. The tumour may then be dealt with in the customary way. Dr. Walton describes this operation only. He has found it the most satisfactory of all. There is, however, no really satisfactory operation for the hypophysis. Comparatively few surgeons have attempted to exploit this department of surgery. Dr. Walton mentions only three patients on whom he has operated. One died eight months after operation, another showed no improvement and the third was considerably eased of some of her symptoms. Other methods have been practised by other surgeons with equally disappointing results. No surgeon has yet demonstrated any impressive series of cases. Cushing has given his *imprimatur* to the temporal route, which was first suggested by Horsley, while the Germans and Americans have largely practised the nasal route of approach through the sphenoid bone. All methods have their high rates of mortality and their failures to produce good results. Perhaps the surgery of this fascinating subject will only be perfected when its physiology becomes more definitely known.

BOYLSTON MEDICAL PRIZES.

It is announced that the Boylston Medical Prize for 1920 has been awarded to Mr. Stuart Mudd, of Saint Louis, United States of America. for his essay on acute inflammation of the nose, pharynx and tonsils.

A prize of five hundred dollars and the Boylston Prize Medal for 1922 is offered for the best dissertation on the results of original research in medicine, the subject to be chosen by the writer. The essays must be in the hands of the Secretary of the Boylston Medical Committee, Dr. H. C. Ernst, Harvard Medical School, Boston, Massachussetts, United States of America, on or before December 31, 1922. Each essay must bear in the place of the author's name, some sentence or device and must be accompanied by a sealed packet, bearing the same sentence or device and containing within the name and address of the author. Any clue by which the authorship of a dissertation is made known to the Committee, will debar the author from the competition. The essays must be printed or typewritten and the pages bound in book form. Further particulars can be obtained on request, addressed to the Editor.

POST-GRADUATE COURSE IN MELBOURNE.

Arrangements have been made for the next session of the Post-Graduate Course to commence on November 7, 1921. Professor W. Agar, F.R.S., will deliver the inaugural address on heredity and Mendelism. A special course of demonstrations in eye and ear work will be included in the programme. Full particulars will be published at an early date

Abstracts from Current Medical Literature.

PHYSIOLOGY.

(122) Gastric Response to Water.

G. F. Sutherland (*Amer. Journ. Physiol.*, March, 1921) has investigated the stimulating action of water on the gastric secretion. The attempt was made to eliminate the effect of psychic influence on the secretion by using dogs with a Heidenhain pouch. The water was given in one of three ways, *viz.*, intravenously, through a gastric fistula and by means of a stomach tube. Water injected intravenously increased gastric secretion. The ingestion of water dilutes the salts of the blood, but it is not thought that the increased secretion was due to this cause, for injection of isotonic and hypertonic salt solutions produced a similar response. There was a continuous activity of the gastric glands and the increase in available fluid in the blood stream hastened the passage of water from the blood to the secreting cells of the gastric glands. The flow of the gastric juice was much greater if the gastric glands were in an active condition at the time of injection. Water introduced *per rectum* produced no effect, but 100 c.cm. of water introduced directly into the small intestine 22 hours after the last meal, caused an increase in gastric secretion which lasted 10 hours. In this case the water might be absorbed rapidly enough to increase the fluid in the blood, or it might act directly through the agency of hormones in the duodenal mucosa, or indirectly through its effect on the absorption of secretagogues from the lumen of the tract. Water introduced into the stomach by means of a stomach tube, stimulated gastric secretion more than the same amount introduced directly into the small intestine. A standard meal consisting of 310 grammes of lean meat was given to a dog. After the maximum rate of digestion secretion had passed, 105 c.cm. of water were given through a stomach tube. The rate then increased above the maximum on the meal alone. The effect of the water on the gastric secretion diminished in proportion to the period of time elapsing between the meal and the giving of water. The longer the period that the water remained in the stomach, the greater the secretory response of the stomach to water. Henderson considers that the giving of food with water would hasten the digestion and absorption of proteins and, in addition, would keep the water in the stomach for a longer period. Water is probably not absorbed from the stomach rapidly enough to stimulate gastric secretion by dilution of the blood. The stimulating effect of water on gastric secretion was greatest when water was injected into the stomach. The effect was more marked after injection of the water into the small intestine than after intravenous injection, and in all cases was greater if food were being digested.

(123) Breathing of Air of Low Oxygen Pressure.

The data collected from the official altitude examinations of aviators in the United States Army have been analysed by E. C. Schneider and D. Truesdell (*Amer. Journ. Physiol.*, March, 1921). The examinations comprised low oxygen tests. The aviator re-breathed from a bag containing 52 litres of air from which the carbon dioxide was removed by passing it over sodium hydroxide, so that there was a progressive diminution in the percentage of oxygen in the air breathed. The pulse rate and arterial pressure, both systolic and diastolic, were determined each minute and the respiration was recorded on a kymograph. Throughout the test the aviator was busy with work directed by a psychologist, whereby the condition of voluntary co-ordination and attention was observed. The degree of oxygen deficiency tolerated showed a wide range. The mean of 2,000 cases was 7.42% of oxygen and the mean duration of the test was 24 minutes. The subjects were classified as fainting and non-fainting types. A good subject who did not faint, might fail as the oxygen tension diminished to attend to first one, then another of the signals directed by the psychologist. He would have inattentive eyes, would be deaf and irresponsive to questions and yet when revived with fresh air, he might ask why he was not carried further. An analysis of 300 subjects showed that 46.7% belonged to the fainting and 53.3% to the non-fainting type. The compensatory factor of first importance was the increase in the volume of the air inspired each minute. The circulatory changes were a secondary factor. There was an increase in pulse rate in all cases. Men of the non-fainting type showed a greater increase in the amount of air breathed during a minute. They had a greater rise in the systolic pressure and a more moderate fall in the diastolic pressure than did those of the characteristic fainting type.

(124) Alveolar Carbon Dioxide Pressure.

E. C. Dodds (*Journ. Physiol.*, March, 1921) finds that in a series of normal persons, the alveolar carbon dioxide pressure shows the following changes after a meal: (*i.*) a rise of from two to six millimetres within the first half or three-quarters of an hour; (*ii.*) a subsequent fall of about the same amount (two to six millimetres) below the original level; (*iii.*) a return to the original level. In a man from whom the greater part of the stomach had been removed for severe gastric ulceration, the rise after a meal was very small in amount (0.4 to 0.8 mm.), while the subsequent changes were similar to those seen in a normal person. This suggests very strongly that the rise seen in a normal person is associated with the secretion of gastric juice. This subject showed the fall of CO_2 alveolar pressure, followed by the return to the original level in just the same way as a normal person. This affords strong evidence that these changes are associated with digestive processes in portions of the alimentary canal below the stomach. Observations made when no food was taken throughout show an almost constant carbon dioxide alveolar pressure. The later changes in alveolar carbon dioxide pressure after a meal may throw light on processes such as pancreatic secretion, which are less accessible to investigation.

(125) Influence of Water Ingestion on Blood Pressure.

J. B. Orr (*Proc. Physiol. Society*, June, 1920) finds that, after three litres of water extra were drunk on one day, there is a fall which is variable in extent in both systolic and diastolic blood pressures after the water has passed through the system. Readings were taken the day before the extra water was taken, the same day and the first day following. The evidence is reviewed that certain pressor substances formed by bacterial action on protein are absorbed from the bowel and the possibility that pressor substances may originate in the tissues from perverted metabolism. It is considered that increased ingestion of water eliminates these pressor substances, thus accounting for the fall in blood pressure. The systolic and diastolic pressures were determined by means of a Riva Rocci instrument with a screw compressor and the auditory method was adopted.

(126) Local Application of Cold.

If a man is seated in a room, the temperature of which is 38° C., droplets of sweat form on his forehead. If he allows a stream of cold water to play over his hands, the sweating ceases. Leonard Hill (*Journ. Physiol.*, March, 1921) has proved, by stopping the circulation in the hands before placing them under the tap, that this is not a reflex nervous effect, but is due to cooling of the blood. The sweating did not stop. Other experiments showed how effectively the heating or cooling of a small portion of the body can heat or cool the whole. In one experiment the heat loss was 72 kilocalories an hour—the total heat production of a resting man.

BIOLOGICAL CHEMISTRY.

(127) Antiscorbutic Powers of Dried Milks.

E. B. Hart, H. Steenbock and N. R. Ellis have determined the presence or absence of the antiscorbutic vitamin in a number of specimens of dried milk prepared by different processes (*Journ. Biol. Chemistry*, April, 1921). Three methods are followed in the United States in the manufacture of dried milk: (*a*) the Merrell-Soule or spray process; (*b*) the spray process developed by the California Central Creameries, in which the milk is not condensed before desiccation and in which the powder is cooled and removed in a few seconds; and (*b*) the Just process,

involving desiccation upon a heated roller. The investigation has been made with guinea-pigs fed on a basal ration of heated alfalfa flour, rolled oats and common salt. The alfalfa flour has been put into an autoclave, heated to 6.8 kilos pressure, for thirty minutes. In most experiments the milk powder has been added in the proportion of 40% of the ration. As the guinea-pigs have eaten about 20 gm. *per diem*, this corresponds to a consumption of 80 c.cm. of fresh milk. Some preliminary experiments have shown that this amount of liquid milk is ample to prevent the onset of scurvy in a guinea-pig. The investigation has shown that dried milk prepared by the spray processes are unable to protect against scurvy in the quantities employed. Whether skim or whole milk has been utilized in the manufacture, the result is the same. Even when the proportion of dried milk in the ration has been raised to 60% by weight, scurvy has occurred after several weeks. The use of dried milk made by the Just method leads, however, to a different milk. The inclusion of 40% dried milk in the ration is sufficient to ward off scurvy. The authors, however, point out that the initial amount of antiscorbutic vitamine in a sample of milk is of importance in judging the value of dried milks. Milk obtained during winter is less potent in warding off scurvy from guinea-pigs than that collected in summer. When the diet of the cows in winter is selected in respect to suitable roots and tubers, the milk becomes richer in antiscorbutic potency. The presence or absence of fats in the milk is without influence on the antiscorbutic properties.

(128) The Stability of Antiscorbutic Vitamins.

N. R. Ellis, H. Steenbock and E. B. Hart have made some experiments on the behaviour of the antiscorbutic vitamins in cabbage and orange juice as a result of the application of different methods of treatment (*Journ. Biol. Chemistry*, April, 1921). The diet used in the investigation has been composed of 69 parts of rolled oats, 25 parts of alfalfa hay which has been treated in an autoclave for thirty minutes at 15 pounds pressure, 5 parts of casein and 1 part of common salt. These substances furnish a ration which satisfies all the factors of nutrition in the guinea-pig, with the exception of the antiscorbutic hormone. In the investigation the various treated materials have been added to the basal ration. The results obtained in the investigation show that cabbage, dried with an atmosphere of carbon dioxide in a vacuum oven at 65° C., has no protective powers against scurvy. The absence of oxygen during desiccation has not prevented the disappearance of the antiscorbutic vitamins. The addition of one cubic centimetre of orange juice to the daily ration has been found sufficient to prevent the appearance of any signs of scurvy in these experiments. This amount has been taken as the minimum for complete protection from scurvy. The previous extraction of the orange juice by shaking it with ether does not diminish the potency, the extracted orange juice possessing equal antiscorbutic powers to untreated orange juice. The aeration of orange juice by bubbling air through it in an aeration bottle has led to no diminution in the antiscorbutic vitamins. The addition of oxidizing agents, such as hydrogen peroxide and potassium permanganate, has caused more or less rapid destruction of the antiscorbutic powers of orange juice. Blood charcoal adsorbs a considerable proportion of the antiscorbutic vitamins of orange juice. The authors have been unable to confirm the result of Harden and Zilva that antiscorbutic vitamins pass unaltered through Chamberlain porcelain candles. They have found that a proportion of the vitamins has been retained by the filter. The guinea-pigs receiving filtered orange juice have shown symptoms of scurvy after ten weeks. They have not, however, succeeded in finding a candle in which the pores have been sufficiently fine to retain the whole of the antiscorbutic vitamin of orange juice.

(129) Fat Metabolism in Diabetes Mellitus.

N. F. Blau and S. T. Nicholson have made an analytical study of the sugar, fat and cholesterol in the blood of 34 patients suffering from diabetes, noting the relationship of these substances to each other and to the diet (*Archives of Internal Medicine*, September, 1920). The patients were all placed on the Allen-Joslin dietetic treatment of diabetes. Specimens of blood were thereafter analysed for sugar, total lipoids and cholesterol content of whole blood and plasma at frequent intervals. From the laboratory findings the cases seemed to fall into three groups. In the first group the blood fat increased steadily or intermittently while the sugar decreased. In the second group the variations in blood fat paralleled those of blood sugar. In the last group the blood fat remained at practically the same level while the blood sugar decreased. Comparison with the clinical records showed that the numbers of each group had certain clinical resemblances to each other. Eleven of the patients showed at the beginning of treatment a marked increase in blood lipoids along with a considerable drop in blood sugar. Clinically these all suffered from severe diabetes. In the case of seven of the patients the blood fats seemed to rise and fall with the blood sugar, decrease in the fat apparently progressing more slowly than the decrease in the blood sugar. All but two of these patients were in a milder stage of the disease. In the case of eight patients there was an almost constant value for the blood lipoids at high level while the blood sugar decreased. They all presented a history of infection prior to the onset of diabetes and suffered from a somewhat more severe form of the disease than those of the preceding group. The diabetic organism can utilize fats more readily in the presence of large amounts of sugar in the body and in sugar-free patients there must be a great strain on the fat burning mechanism when large amounts of fat are given as food.

(130) Carbon Dioxide in Blood.

J. B. Collip (*Journ. Biol. Chemistry*, March, 1921) has measured the amount of carbon dioxide liberated from blood, serum and plasma which have been exposed to carbon dioxide of different tensions. Although it had been accepted as proved for many years that the carbon dioxide borne by the blood is carried mainly in the plasma, certain observations have recently suggested that a considerable proportion may be carried in combination with proteins in the red blood corpuscles. The author has conducted this series of experiments to show that the exposure of whole blood to carbon dioxide leads to a diminution of the quantity of chlorides in the plasma and to an augmentation in the amount of bicarbonate. He has also made some experiments to show that the shaking of blood with ordinary air leads to the passage of chlorides from the red blood corpuscles to the plasma and to a diminution of the amount of bicarbonates in the blood. The author considers that his results support the contention that sodium bicarbonate, rather than combinations of carbon dioxide with protein, transports the carbon dioxide in the blood. The author has also shown that solutions of the ash of serum or of whole blood have a greater power of combining with carbon dioxide than the original serum or whole blood. From this fact the author deduces that a portion of the base in the blood is bound to an organic substance (probably proteins) which behaves as a weak acid.

(131) Size of Fat Globules in Milk.

E. W. Schultz and L. K. Chander (*Journ. Biol. Chemistry*, March, 1921) have measured the diameters of the fat globules in 45 samples of goats' milk. A dark ground illuminator has been employed on the microscope. The specimens of milk have been mounted as capillary films between a slide and cover-slip and the cover-slip has been sealed with vaseline. Forty-five samples of milk from fifteen goats have been studied. The average percentages of fat globules of different sizes in the 45 samples have been: below 2 μ, 57%; between 2 μ and 4 μ, 34%; between 4 μ and 6 μ, 7%; between 6 μ and 8 μ, 2%; and above 8 μ, none. Ninety-one *per centum* of the fat globules are under four microns in diameter. In cow's milk over 90% of the fat globules are over four microns in diameter and 40% of the fat globules are over twelve microns in diameter. In human milk the fat globules are still larger. The authors consider that the greater surface area of the fat globules in goats' milk should render the fat more accessible to the lipase of digestive juices and that the fat of goats' milk should be more rapidly and completely digested than that of cows' milk.

British Medical Association News.

SCIENTIFIC.

A meeting of the Western Australian Branch was held at the Perth Public Hospital on April 20, 1921, Dr. G. W. Barber, C.B., C.M.G., D.S.O., the President, in the chair.

Dr. M. Kasner Moss read a paper on anæsthetics and their administration (see *The Medical Journal of Australia*, August 6, 1921, page 96).

Dr. J. Bentley, M.C., stated that he had given ethyl chloride-ether sequence. He held that ethyl chloride given alone in a closed inhaler was dangerous, especially if a second administration were attempted.

Dr. T. L. Anderson, O.B.E., agreed with the last speaker concerning the danger of a repeated administration of ethyl chloride in a closed inhaler. He spoke in favour of the use of rectal anæsthesia in operations on the larynx.

Dr. F. Gill dealt with rectal anæsthesia and described the method in use at the West London Hospital. The patients were kept in the hospital for a week prior to the operation for the purpose of preparation. He considered it an excellent method for certain operations.

Dr. K. G. McK. Aberdeen said that he had used rectal anæsthesia in between 200 and 250 cases, first at the Women's Hospital. The dose of ether was calculated on the following basis: For women, 3.1 c.cm. was given per kilogram body weight; for men, 3.48 per kilogram; for patients with septic infections, from 2.0 and 2.5 c.cm. per kilogram; and for children, from 6.25 to 7.8 c.cm. per kilogram. Olive oil (25%) was dissolved in the ether (75%). In administering the ether to children it was often difficult to gauge the correct dose, especially when there was a cleft palate. The absorption in children was slow. An enema was given both on the night before and on the morning of the day of the operation. A dose of one gramme of chloretone in salad oil was given to adults one and a half hours before the operation to diminish the irritability of the bowel. From three quarters to one hour before the operation, a catheter fitted with a rubber tube and funnel was passed into the rectum and the ether-olive oil solution was introduced. Dr. Aberdeen stated that the bowel should be washed out immediately after the operation, or better, the *sphincter ani* should be dilated before the patient left the table and when he arrived in the ward, the bowel washed out. If collapse occurred, the only treatment of value was the injection into the heart of from 12 to 24 milligrammes of atropine. For nervous children he advocated a small dose of morphine hypodermically one hour before the operation.

Dr. G. W. Barber stated that the nitrous oxide-oxygen-ether admixture had proved a very valuable agent in the field.

J. K. Couch gave some details of a death under ethyl chloride administered with a closed inhaler. The patient had died within about two minutes. In his experience induction with the Shipway inhaler was too slow; he used a little chloroform at the start. He frequently used Clover's inhaler for ether anæsthesia and usually completed the induction stage in from four to five minutes.

Dr. J. J. Holland spoke of a patient who had died on the table during the administration of ether. The patient had been very anæmic from prolonged hæmorrhages. He held that the death could not be ascribed to the anæsthetic.

Dr. W. Trethowan said that in his early days chloroform was used exclusively. The Scotch method of slow induction and uniform administration made for safe anæsthesia. He asserted that these were essential factors for safety. There had been a tendency to use excessive quantities of chloroform. He expressed the opinion that ether was not a safe anæsthetic for elderly people, on account of its action in causing pulmonary irritation.

Dr. R. C. Merryweather was strongly in favour of hypodermic injections of morphine and atropine given one hour before the operatiin when ether was administered. He claimed that it diminished the post-operative shock and vomiting. He used Probyn Williams's apparatus. He had been taught that slow induction with chloroform was more dangerous than the more rapid method. He suggested that the fees for anæsthesia were inadequate. This question should be considered. In his opinion oxygen and ether was the ideal anæsthetic mixture.

Dr. D. P. Clement said that he did not agree with Dr. Trethowan in regard to the administration of chloroform. Chloroform was a cardiac depressant. The warning of impending danger recognizable when ether was employed, was absent in the case of chloroform. He strongly advised younger practitioners to use ether exclusively. When he employed ethyl chloride for induction prior to ether anæsthesia, he stopped the ethyl chloride just before the snoring stage was reached. Chloroform was used regularly at the Rotunda Hospital; it was quite suitable for midwifery practice, as deep anæsthesia was not required.

In his reply, Dr. M. K. Moss said that there had been one death at the Base Hospital at Caulfield, when rectal anæsthesia had been used. In regard to the hypodermic injection of atropine into the heart as a means of resuscitation, he held the opinion that the actual puncture of the heart and not the drug produced the good effect. It had been stated that auricular fibrillation occurred at times during slow induction of light anæsthesia. The objection to a preliminary injection of morphine and atropine was that as it caused a pin point contraction of the pupil, it became difficult to determine the exact degree of anæsthesia.

A meeting of the Western Australian Branch was held at the Perth Public Hospital on May 18, 1921, Dr. G. W. Barber, C.B., C.M.G., D.S.O., the President, in the chair.

Mr. D. M. McWhae, C.M.G., C.B.E., presented two patients and read the notes concerning them. The first patient was a woman, aged 21 years, who walked with gross ataxia. Her gait resembled that of a drunken man rather than the stamping gait of a patient with *tabes dorsalis*. The staggering became more marked at night time than in daylight. There was definite Rombergism. The knee jerks and ankle jerks were absent. Dr. McWhae raised the question whether she was suffering from the juvenile form of *tabes dorsalis*, which was a very rare condition. He pointed out that her pupils reacted both to light and to accommodation. The other physical signs appeared to exclude *tabes dorsalis*. There was a distinct intention tremor of the arms. This was elicited when she attempted to pick up anything. She had difficulty in lacing her shoes and in tying the laces. Her speech was syllabic or scanning. Nystagmus was present when she turned her eyes to the extreme right. Babinski's plantar reflex was obtained in each foot. There was some loss of control over the bladder. She lost her temper on slight provocation and was easily moved to tears. The psychical symptoms and physical signs were characteristic of disseminated sclerosis. Dr. McWhae, however, had concluded that this was not the correct diagnosis. He found that she was suffering from Friedreich's ataxia. There was lordosis with some scoliosis, commencing *pes cavus* and the deformity of the great toe typical of Friedreich's ataxia. The onset of the affection occurred when she was ten years of age. From the outset there had been ataxia; the change in the gait had been progressive and she was at the time of the meeting able to walk with very considerable difficulty. He thought that considerable benefit should accrue as a result of Fränkel's exercises and re-education. He had not been able to investigate the whole family history, but, as far as he had been able to ascertain, none of her near relatives had suffered in a similar manner.

Dr. McWhae's second patient was a soldier, aged 28 years, who six years before had had a white skin. From that time his skin had gradually become pigmented until the integument of his face and arms was very dark. The skin of his body was also pigmented, but to a less degree. Although there was no pigmentation of the mucous membrane of the mouth, Addison's disease had been suggested; his symptoms had been chiefly gastro-intestinal, which was characteristic of that disease. Every ten to fourteen days the patient had had attacks of vomiting and diarrhœa. Asthenia had been completely absent. Dr. McWhae held that the diagnosis of Addison's diseases in the absence of asthenia was not justified. Moreover, his blood pressure was not low, the systolic pressure being 170 mm. and the diastolic 110 mm. of mercury. Careful inquiry had failed to elicit the use of drugs, such as silver, arsenic or carbolic acid, which might have caused the pigmentation. The disease was certainly not ochronosis, in which there was blackening of the cartilage, a bluish-black discoloration of the ears and deep pigmentation

of various regions of the skin. This condition was due to carbolic acid or alkapton; there was no alkapton in the urine of the patient. Dr. McWhae explained that alkapton caused the urine to turn black after it had been passed. A transient blue colour appeared on the addition of a weak solution of ferric chloride. In the next place he was able to exclude chronic jaundice, as the sclera was quite clear. He believed that the disease was hæmochromatosis. Up to the year 1911 only 60 cases of this disease had been recorded. The diagnosis was made on the association of pigmentation with the signs of cirrhosis of the liver and fibrosis of the spleen and pancreas. In 1916 the patient had been admitted to hospital. At that time the spleen and liver were found to be greatly enlarged. Since then both had diminished in size. In November, 1920, an abdominal operation had been performed; it was then ascertained that the liver was cirrhosed and divided into lobules by deep fibrous bands. The pancreas was also very firm. There was evidence of pancreatic insufficiency. Creatorrhœa was present. This could be determined by the finding of great numbers of undigested muscle fibres in the fæces after a meal of under-cooked meat and of an increase of diastase in the urine. Normally one cubic centimetre of urine contained enough diastase to digest 20 c.cm. of a 0.1% solution of starch in half an hour, *i.e.*, 20 units. One cubic centimetre of the patient's urine digested 100 c.cm. of the starch solution, *i.e.*, 100 units. In hæmochromatosis there was no response to Loewi's adrenalin test and glycosuria was not found. Some pathologists ascribed the pigmentation to hæmolysis; others held that the disease was a primary disorder of metabolism with changes in the chromogenic portion of the protein molecule and deposition of pigment. The pigment was deposited in the viscera, the liver, pancreas, spleen, etc., and in the skin. In the later stages the islands of Langerhans in the pancreas were usually involved and glycosuria resulted. The condition was then spoken of as bronzed diabetes. It was a severe diabetes and frequently led to coma. This patient's blood serum had been subjected to the Wassermann test and an incomplete reaction had been obtained. He had been treated with galyl and large doses of potassium iodide.

Dr. J. J. Holland presented a Chinaman, aged 50 years, with a palpable aneurysm to the right of the sternum in the region of the second and third ribs. There was a distinct impulse on palpation. The patient had not had any pain. His occupation involved the pushing of a hand barrow.

Dr. F. Gill showed a boy aged 15 years, with an undiagnosed skin eruption.

Dr. D. P. Clement read some notes of an interesting obstetric condition.

Dr. A. Juett read a paper on muscular atrophy (see page 213).

Dr. G. W. Barber thanked Dr. Juett for his valuable paper. He said that experience gained in war surgery had been of great benefit in the treatment of nerve and muscle injuries.

Dr. E. E. Moule asked for advice in regard to the prevention of the morbid conditions of which Dr. Juett has spoken.

Dr. D. D. Paton asked Dr. Juett whether he advocated quinine ionization.

Dr. M. K. Moss wished for information concerning the results of nerve grafting.

In his reply Dr. Juett stated that it was difficult to give general advice concerning prevention. In the case of synovitis of the knee joint, after the swelling had been reduced, attention should be directed to the *quadriceps femoris*. Callipers should be applied and muscle exercise should be allowed only while the patient was lying in bed. Small, frequent contractions could be used. Later gradual flexion of the knee should be effected. Lateral movements of the patella were liable to lead to a relapse of the synovitis.

In speaking of ionization, Dr. Juett stated that the penetration of the ions varied with the amount of current and the depth of the tissues. Quinine ions left the positive pole and travelled toward the negative pole. The ions certainly penetrated tissues.

Nerve grafting for poliomyelitic lesions, as a rule, did not yield good functional results.

Herbert Giblin Furnell, Esq., M.B., B.S. (1921, Univ. Melb.), of St. Vincent's Hospital, Melbourne, and William Anselm Collopy, of 7 Waltham Street, Richmond, have been elected members of the Victorian Branch.

Clifton John Barry Armstrong, Esq., M.B., Ch.M., 1921 (University of Sydney), of Sydney Hospital, has been nominated for election as a member of the New South Wales Branch.

A PROPOSED PÆDIATRIC SECTION.

A meeting of medical practitioners interested in pædiatrics was held in the Library of the British Medical Association Building, 30 to 34 Elizabeth Street, Sydney, on September 6, 1921, for the purpose of discussing the proposal to establish a section of pædiatrics of the New South Wales Branch of the British Medical Association.

The following resolutions were adopted by a unanimous vote:

(*i.*) That this meeting strongly favours the formation of a pædiatric section of the New South Wales Branch of the British Medical Association and requests the sanction of the Council thereto, in conformity with the Articles of Association regulating sections for special branches of medical knowledge.

(*ii.*) That the office-bearers of the proposed section consist of a chairman, vice-chairman, honorary secretaries and honorary treasurer.

(*iii.*) That the following are elected as temporary or provisional office-bearers, to be confirmed later after sanction has been obtained:

Chairman: Dr. C. B. Clubbe.

Vice-Chairmen: Drs. J. Macdonald Gill, W. F. Litchfield, Professor A. E. Mills.

Members of Committee: Drs. W. G. Armstrong, Margaret Harper, E. S. Littlejohn, J. S. Purdy, Harvey Sutton, R. B. Wade.

Joint Honorary Secretaries: Dr. Selwyn Harrison, Dr. Wilfred Vickers.

Honorary Treasurer: Dr. Edgar Stephen.

(*iv.*) That the annual subscription be ten shillings.

A general meeting will be announced after the approval of the Council of the New South Wales Branch has been received. The co-operation of all the members of the Branch is cordially invited in order to make the section an assured success.

Medical Societies.

(Affiliated with the British Medical Association.)

THE NORTH-EASTERN MEDICAL ASSOCIATION (NEW SOUTH WALES).

The annual meeting of the North-Eastern Medical Association of New South Wales was held at the Freemason's Hall, Lismore, on May 11, 1921, Dr. P. Corlis in the chair.

The ordinary business concerning applications by members to be permitted to accept appointments as medical officers to friendly society lodges was dealt with.

Dr. R. S. R. Mitchell and D. A. W. Raymond, both of Maclean, were elected members.

The Honorary Treasurer presented the financial statement and balance sheet. On the motion of Dr. J. I. Robertson, seconded by Dr. T. W. Van Epen, the documents were received.

Dr. P. Corlis gave notice that he would move at the next general meeting:

That this Association consider the question of drawing up a schedule of fees for practitioners within this area.

Dr. O. A. Diethelm moved and Dr. A. J. Opie seconded that the President, the Vice-President and the Honorary Secretary be appointed a sub-committee to consider the schedule and to circularize the members with the object of embodying local rates of fees in the schedule.

The following were elected office-bearers and members of the Committee for the ensuing year:

President: Dr. A. J. Cahill.

Vice-President: Dr. J. I. Robertson.

Honorary Secretary and Honorary Treasurer: Dr. R. V. Graham.

Honorary Auditor: Dr. A. M. Aspinall.

Members of the Committee: Drs. R. Belli, C. C. Corlis, P. Corlis, O. A. A. Diethelm, T. G. C. Evans, D. D. Gibson, J. Goldsmid, G. W. Macartney and A. J. Opie.
Delegates: Dr. O. A. A. Diethelm, Dr. T. Henry.

Analytical Department.

The Analytical Department of *The Medical Journal of Australia* has been established for the purpose of presenting to the medical profession a record of a careful inquiry into the manufacture and ingredients of food products and proprietary preparations. In each case a specially selected inspector has been engaged to carry out an exhaustive inspection of the preparation of the article. The inspector has collected samples of ingredients and of the finished product for analysis. For comparison samples are also purchased in the open market and analysed side by side with the manufacturer's samples. The whole cost of inspection and of analysis is borne by the *Journal*. Moreover, no manufacturer is asked to advertise his products prior to the completion of the inspection and analysis and the publication of the report. Every care is taken to ascertain all the relevant facts concerning these preparations, so that the opinions expressed in this column can be accepted by our readers as authoritative and may be used as guides in practice. While the reports are published editorially and while the whole responsibility for them is borne by the Editor, it must be pointed out that they represent the weighed verdicts of experts, controlled in special instances by consultation with dieticians, pharmacologists, clinicians or chemists. As has been announced, all food preparations and proprietary remedies manufactured in Australia accepted for advertisement by *The Medical Journal of Australia* must be subjected to this inquiry and found to be carefully made from pure ingredients, to be suitable for the conditions for which they are to be used and to be of a high standard. For the present it is not intended to include pure chemicals or bacteriological products in this undertaking. The scheme cannot be applied suddenly, as many advertisers still have unexpired contracts which cannot be disturbed. The work of inspection and analysis is proceeding as rapidly as circumstances will allow.

HEXSOTATE.

Hexostate is an acetate of hexamine, formerly known as urotropine. It is manufactured by Messrs. Felton, Grimwade & Company, of Melbourne. The manufacturers claim that it is an internal disinfectant and antiseptic and that it acts not only in the presence of acid, but also when the medium is alkaline.

Manufacture.—The ingredients employed in the preparation are hexamine of English origin, caustic soda, also imported from England, and acetic acid of Australian manufacture. The first stage of the preparation is the formation of an alkaline sodium acetate solution, which has to be filtered. This solution is mixed with the hexamine in definite proportions. The resulting clear solution is evaporated with the aid of gentle heat to a cake and the mass is broken by a tooth mill to a powder. The powder is triturated in a ball mill to a state of very fine division. It is then passed through a granulator and thence to the tablet machine.

Our inspector informs us that he is quite satisfied that the process is conducted throughout with proper attention to order and cleanliness. The rooms, containers, instruments and implements are kept in a clean state and the operators' hands and clothes are also clean. Impurities could not enter in the powder, save from the copper vacuum pan. A trace of copper would become manifest by a change of the pure whiteness of the product. The hexamine seen at the factory was of a uniformly white crystalline structure.

Precautions seem to be taken to prevent the possibility of the Hexsotate tablet machine being fed with a different product. These precautions include an elaborate system of labelling of every chemical, a special device whereby poisonous substances are worked up on a set day, a system of sampling and analysis by a trained chemist and, lastly, the adjustment of the machine to the consistency of Hexsotate.

From the methods employed in the manufacture, it is improbable that any deterioration or alteration of the active ingredients takes place in the finished product. Our inspector concludes that the manufacture of Hexsotate is completely satisfactory from the point of view of the proper conduct of the processes involved.

Analysis.—Samples of the hexamine—$(CH_2)_6N_4$—used in the manufacture yielded 39.6% nitrogen and 51.15% carbon (average), which when calculated to hexamine would yield 99% and 99.1%. There was 1.05% of moisture. No sulphates, chlorides, heavy metals or para-formaldehydes were detected.

A sample of sodium acetate was also analysed. The solution (1.1%) contained 0.8% of iron, estimated as Fe_2O_3. There was 0.2% sodium sulphate and 0.5% sodium chloride.

The analysis of samples of Hexsotate tablets showed that the bought tablets compared satisfactorily with those collected at the factory. Each tablet contained 0.97 gramme. The result of the analyses is as follows:

Hexamine	32.05%	(Average)
Sodium acetate	43.00%	
Moisture	24.60%	
Iron (Fe_2O_3)	0.20%	
Chloride	Trace	
Sulphate	Trace	

No trace of any of the heavy metals was detected. The analyst failed to obtain evidence that Hexsotate undergoes dissociation by hydrolysis in an alkaline solution more readily than does hexamine in an alkaline solution. In the human body, as in the test tube, hexamine (urotropine) yields very little formaldehyde except in an acid fluid. Its therapeutic uses are recognized as being limited to its action in the bladder, provided that the urine is distinctly acid in reaction. The employment of the acetate or the citrate is advocated on account of the alleged activity in an alkaline medium. If therapeutic experience reveals a more ready hydrolysis within the organism of the acetate, Hexsotate would certainly represent a more useful drug than hexamine. The purity of the preparation and the care with which it is handled justify us in stating that it can be recommended in all conditions in which hexamine (urotropine) is indicated.

Naval and Military.

APPOINTMENTS.

The following appointments to substantive commissioners on the Reserve of Officers have been promulgated in the *Commonwealth of Australia Gazette*, No. 70, of September 8, 1921:

Australian Military Forces.

Second Military District.

The Undermentioned Honorary Major to be Lieutenant-Colonel—
George Raffan.

To be Major—
Honorary Major William Chisholm, Australian Army Medical Corps Reserve.

The Undermentioned Honorary Captains to be Majors—
Harold Octavius Lethbridge, M.B.E., Charles Herbert Wesley, Edwin Charles Temple Smith, George Lawson Kerr, John Thomas Jones, M.C., Stanley Roy Stafford and Fritz Peter Max Solling, M.C..

The Undermentioned Honorary Captains to be Captains—
George Bell, O.B.E., Alan Neil Yuille, Stanley Vincent O'Regan, M.C., Allan Melrose Purves, M.C., and Charles Badham.

To be Captains—
Honorary Captains Ivie Aird, Jack McFadzean Rossell and Robert Murrell Allport, Australian Army Medical Corps Reserve.

Third Military District.

The Undermentioned Honorary Major to be Major—
Percy Samuel Webster.

To be Majors—
Honorary Captains Harold Alfred Cardale Irving and

Edward Elmslie Brown, Australian Army Medical Corps Reserve.

The Undermentioned Honorary Captains to be Captains—
Henry Horwick Holland, John Samuel McLean, Ralph Harry Crisp, Harold Rupert Hyett, Olaf Francis de Lacy, Colin Friend and William Joseph Newing.

To be Captains—
Honorary Captains Henry Fitzgerald Maudsley, M.C., Alan Bothwell McCutcheon, Harold Clive Disher and Eric Neville Harle Gandevia, Australian Army Medical Corps Reserve.

Fourth Military District.

The Undermentioned Honorary Captain to be Captain—
Brian Formby Moore.

Fifth Military District.

To be Captain—
Honorary Captain William Saunders Myles.

Sixth Military District.

To be Major—
Honorary Captain Walter Henry Tofft, M.B.E., Australian Army Medical Corps Reserve.

Correspondence.

HEAT LOSS DURING ANÆSTHESIA.

Sir: The subject of loss of temperature during anæsthesia has interested me for some years. In operations on animals provision is always made in physiological laboratories for the maintenance of a good body temperature. Merely warming the air of the room or applying blankets is not considered adequate. Nor are hot water bottles regarded as satisfactory. The whole operating table, in fact, is kept warm artificially, preferably with electric energy. I have been able to keep a dog under deep chloroform anæsthesia for eight hours without any loss of circulatory or reflex tone and I attribute this largely to the precautions taken to safeguard body temperature. Why surgeons have not adopted this device in human operations has always puzzled me. Whether it is the traditional dislike to import the lore of the physiological laboratory into the operating room or whether modern surgical procedures are so expeditiously conducted that the question of the patient's temperature has no practical significance, I cannot say.

I admit, of course, that the dog, cat and rabbit—the animals usually operated on in laboratories—have each a much larger specific surface than human beings and the rate of cooling must be considerably greater.

Dr. Corlette has certainly established his contention that exaggerated breathing of air of low humidity can extract large amounts of heat from the respiratory tract and hence from the body; but I venture to think that one important factor has been omitted from his calculations. This factor is the variable heat supply within the body. As is well known, the muscles, even during rest in the recumbent position, produce large amounts of heat. for they maintain their tone, which, though it does not display the active metabolism of muscular contraction giving movement, is still in an excited state producing no actual work and giving heat as the sole result of the chemical changes within. That this is the case is seen when the muscles of an anæsthetized dog are paralysed with curare. Here, unless heat is supplied abundantly from without, the temperature of the animal at once begins to sink until it is near that of the air of the room.

I have observed in non-curarized animals where, owing to vagotomy, I have supplied artificial respiration that the tendency to fall in temperature is greater when the animal is deeply under (chloroform) than when the anæsthesia is light, the ventilation of the lung remaining the same. Theory would indicate that the greater the flaccidity of the muscles the less is the heat produced in them and hence the greater the tendency of the body temperature to fall. While I cannot give a quantitative assessment of the heat produced in the manner that Dr. Corlette has done so interestingly with the heat loss, I still think that this is a factor too large to be overlooked and may actually be of greater significance in its effect upon body temperature.

Yours, etc.,
W. A. OSBORNE.

University of Melbourne,
September 5, 1921.

THE TREATMENT OF VENEREAL DISEASES.

Sir: In your journal of September 10, 1921, there is a letter on the treatment of venereal disease by an anonymous writer—"Medico."

Anonymous letters which contain serious statements, are most unsatisfactory. The writer should have sufficient faith in his allegations to publish his name at the end of the letter—or not write at all.

To make statements is one thing, but to prove them to be true is frequently quite another matter.

Dealing, as I do, with purely venereal cases, various matters have on several occasions been mentioned to me, but I have never made these matters public because, as I have said before, it is frequently very difficult to prove the truth of one's statements.

I do not agree with "Medico" that "nearly every chemist treats these cases." From hearsay of some of my patients in private and at the hospital clinic I believe that some chemists still treat venereal diseases, but that the *Venereal Act* has stopped the more cautious consulting chemists from carrying on their former practice. A really respectable chemist does not treat venereal disease.

With regard to his other statement (concerning open clinics where treatments are given at 1s. and 2s.) from what I have heard his information may be correct.

Has it occurred though to "Medico" that if he can prove that these open clinics, conducted by unqualified people, exist (which he may be called upon to prove), that the police should then also be aware of their existence and if they are aware of their existence and have taken no steps for their suppresion, what is one to think?

The whole matter is very serious if the *Venereal Diseases Act* is intended to be taken seriously.

These matters, however, can be fully discussed at the meeting of the New South Wales Branch of the British Medical Association on October 29, when members of Parliament interested in the venereal problem could be invited to attend.

I personally believe the *Act* to be unsatisfactory—due in part to lack of organization in the medical profession. We, as a whole, have done but little to combat the venereal problem even from a prophylactic point of view. I wonder is it because we have never tried to pull together on that subject.

Parliament, the Church, and the press appear to take the way of least resistance, as they seem to fear to offend the people. We toddle along apathetically behind them. What does it matter!

The problem is one that many a man would willingly set himself to solve were he only given the opportunity. For him who brings dusk to the long venereal day, much honour waits.

Yours etc.,
J. COOPER BOOTH.

283 Elizabeth Street, Sydney,
September 10, 1921.

THE TEACHING OF OBSTETRICS.

Sir: May I be permitted to endorse most strongly one paragraph in your leader of September 3 inst., *viz.*, "From a purely practical point of view there is urgent need for better and more scientific training in obstetrics." I suppose that at least 90% of students will ultimately become general practitioners and as such the most important branch of their work will be midwifery. Alone, and depending entirely on their own knowledge, manual skill and presence of mind, they will have to carry to a successful issue what may be a simple physiological process or may tax their resources to the utmost. No time for sending for help in a case of *post partum* hæmorrhage, nor often in one of obstructed labour. The importance of an adequate knowledge

of the prevention of puerperal infection has been again shown by the discussion—still in progress—in the columns of the *British Medical Journal*.

I do not know the present regulations, but in my time—thirty-two years ago—a certificate of attendance on twenty labours, with a course of lectures, was all that was necessary to present oneself for examination.

Whether the woman of the present day is differently constituted to her predecessor of the last century I do not propose to discuss, but I know that in the last twenty labours I have attended there have been three unreduced occipito-posterior presentations and one foot presentation, while forceps have been required in seven cases from sheer inability to bring forth the child; and this experience tallies with that of my predecessor in the practice.

Experience should be gained by the student by actual attendance under the guidance and assistance of his teacher, not after qualification.

I would urge strongly that less time be devoted to teaching the future general practitioner operations which he will never perform and diagnosis of obscure nerve diseases which he will never see, and more to the methods of that branch of the healing art which he will inevitably be called on to practise, midwifery and diseases of women.

Yours etc.,

H. W. WEBBER, M.D. and M.S. Lond., F.R.C.S. Edin.

Bellerive, Hobart,

September 8, 1921.

Medical Appointments.

Dr. Namon L. Cass (B.M.A.) has been appointed Retained Medical Officer to the Nannine District Hospital and District Medical Officer at Nannine, Western Australia.

Dr. O. G. Tunks (B.M.A.) has been appointed Government Medical Officer at Coff's Harbour and Dr. W. H. Ward (B.M.A.) at Denman, New South Wales.

Under the provisions of *The Workers Compensation Act*, Dr. R. M. W. Webster (B.M.A.) has been appointed Certifying Medical Practitioner for the Municipal District of Fingal, Tasmania.

It is announced that Dr. D'Arcy Rivers Warren Cowan (B.M.A.) has been appointed Honorary Assistant Physician and Dr. Douglas Lewis Barlow (B.M.A.) Honorary Clinical Pathologist to the Adelaide Hospital Department.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C..

Branch.	APPOINTMENTS.
NEW SOUTH WALES. (Hon. Sec., 30-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIA. (Hon. Sec., Medical Society Hall, East Melbourne.)	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Manchester Unity Independent Order of Oddfellows. Mutual National Provident Club. National Provident Association.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIA. (Hon. Sec., 6 Bank of New South Wales Chambers, St. George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington New Zealand.

Diary for the Month.

Sept. 20.—N.S.W. Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
Sept. 21.—W. Aust. Branch, B.M.A..
Sept. 22.—Clinical Meeting at the Hospital for Sick Children, Brisbane.
Sept. 23.—Q. Branch, B.M.A., Council.
Sept. 28.—Vic. Branch, B.M.A.: Council.
Sept. 29.—S. Aust. Branch, B.M.A..
Sept. 30.—N.S.W. Branch, B.M.A.: Election of two members to Federal Committee.
Sept. 30.—Vic. Branch, B.M.A.: Election of two members to the Federal Committee.
Oct. 1.—Vic. Branch, B.M.A.: Election of Representatives of Divisions.
Oct. 4.—N.S.W. Branch, B.M.A., Council (Quarterly).
Oct. 5.—Vic. Branch, B.M.A..
Oct. 7.—Q. Branch, B.M.A..
Oct. 7.—N.S.W. Branch, B.M.A.: Annual Meeting of Delegates of Local Associations with the Council (First Day).
Oct. 8.—N.S.W. Branch, B.M.A.: Annual Meeting of Delegates of Local Associations with the Council (Second Day).
Oct. 11.—Tas. Branch, B.M.A..
Oct. 12.—Melb. Pædiatric Society (Vic.).

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.

All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building, 30-34 Elizabeth Street, Sydney. (Telephone: B. 4635.)